MEDICAL PROCEEDINGS

MEDIESE BYDRAES

A South African Journal for the Advancement of Medical Science

'n Suid-Afrikaanse Tydskrif vir die Bevordering van die Geneeskunde

P.O. Box 1010 · Johannesburg | Posbus 1010 · Johannesburg

Vol. 4 | 5 April 1958 | No. 7

REDAKSIONEEL · EDITORIAL

DIE DURBANSE MEDIESE SKOOL EN DIE SWAARD VAN DAMOKLES

'n Belangrike debat oor die bestemming van die Durbanse Mediese Skool het plaasgevind op die ses-en-sestigste vergadering van die Suid-Afrikaanse Geneeskundige Raad wat verlede maand in Kaapstad gehou is.

Die debat het voortgespruit uit 'n besluit van die Uitvoerende Komitee om nie getuienis voor die Kommissie insake die Afsonderlike Universiteitsopleiding-wetsontwerp af te lê nie omdat die Uitvoerende Komitee geadviseer is en self die mening huldig dat die oordrag van die Durbanse Mediese Skool nie binne die bestek van die Wetsontwerp val nie.

In die loop van die debat het prof. I. Gordon (dekaan van die Fakulteit van Geneeskunde aan die Universiteit van Natal) voorgestel dat, met die oog op die ontwikkelings wat plaasgevind het sedert die Uitvoerende Komitee sy jongste besluit geneem het, die Geneeskundige Raad getuienis voor die Kommissie moet aflê. Hy het die aandag van die Raad gevestig op 'n verklaring wat op 4 Februarie deur die Edelagbare Waarnemende Minister van Onderwys in die Volksraad gedoen is, nl. dat die Regering van plan is om die Mediese Skool onder die beheer van 'n Staatsdepartement te plaas.

Die debat is beëindig toe die volgende amendement, voorgestel deur dr. Maurice Shapiro (Johannesburg), deur die Raad aanvaar is:

„Dat 'n afvaardiging na die Ministers van Gesondheid en Onderwys gestuur moet word om 'n verduideliking te verkry van die verklaring wat op 4 Februarie 1958 deur die Waarnemende Minister van Onderwys in die Volksraad gedoen is, nl. dat die Regering voornemens is om die beheer van die Durbanse Mediese Skool van die Universi-

THE DURBAN MEDICAL SCHOOL AND THE SWORD OF DAMOCLES

At the sixty-sixth meeting of the South African Medical Council in Cape Town last month, an important debate took place on the fate of the Durban Medical School.

This arose from a resolution of the Executive Committee not to give evidence before the Commission on the Separate University Education Bill, because the Executive Committee had been advised and was of the view that the transfer of the Durban Medical School did not come within the scope of the Bill.

In the debate Prof. I. Gordon (Dean of the Faculty of Medicine in the University of Natal) moved that, because of developments since the Executive Committee had taken its last resolution, the Medical Council should give evidence before the Commission. He drew the attention of the Council to a statement made by the Hon. the Acting Minister of Education on 4 February in the House of Assembly that the Government intended to transfer the Medical School to the control of a State department.

The debate was resolved by the adoption by the Council of the following amendment moved by Dr. Maurice Shapiro (of Johannesburg):

'That a deputation be sent to the Ministers of Health and Education in order to obtain clarification of the statement made by the Acting Minister of Education in the House of Assembly on 4 February 1958 that it was the intention of the Government to transfer the control of the Durban Medical School from the University of Natal to a Department of State. If it is the Government's intention to legislate in this direction under the provisions of the Separate University Education Bill, the deputation shall take the necessary steps to give evidence to the Commission; alternatively,

teit van Natal na 'n Staatsdepartement oor te dra. Indien dit die Regering se plan is om wetgewing in hierdie verband kragtens die bepalings van die Afsonderlike Universiteitsopleidingwetsontwerp in te dien, sal die afvaardiging die nodige stappe doen om getuienis voor die Kommissie af te lê; so nie, indien die Regering voornemens is om wetgewing in te dien om die Durbanse Mediese Skool op 'n ander manier na 'n Staatsdepartement oor te dra, sal die afvaardiging weer eens die Raad se menings in hierdie verband onder die aandag van die Minister bring.'*

In die loop van die debat het prof. Gordon die Raad meegedeel dat op 'n vergadering van die Fakulteit van Geneeskunde wat op 5 Maart 1958 gehou is, 91 van die 92 lede wat teenwoordig was (een het nie gestem nie) opnuut bevestig het dat hulle nie hul onderrigwerk gaan voortsit indien die Mediese Skool van die Universiteit geskei of in 'n staatsbeheerde inrigting omgeskep word nie.

Hy het ook geopenbaar dat die Administrateur-in-Uitvoerende-Komitee van die Natalse Provinsiale Raad besluit het ,dat die Administrasie nie bereid is om 'n ooreenkoms oor die gesamentlike instandhouding van die Mediese Skool met enige onderwysowerheid behalwe die Universiteit van Natal aan te gaan nie. Dit is 'n saak van fundamentele beleid.'

Dit is tans duidelik dat veel meer as die Universiteit van Natal by die saak betrokke is. Die Natalse Provinsiale Administrasie, die Suid-Afrikaanse Geneeskundige Raad en die mediese professie wat die Fakulteit en die onderrighospitaal van die nodige personeellede voorsien, is ewe seer daarmee gemoei. Nie alleen is die verwikkeling van die hele mediese professie in Natal (en miskien ook in die res van die land) nou 'n voldonge feit nie, maar ons staan van aangesig tot aangesig met die onstigtelike vooruitsig dat die Regering geen professionele samewerking van die huidige personeel sal ontvang as hy op die uitvoering van sy voorgestelde plan aandring nie. Dit beteken 'n algehele opvoedkundige *impasse,* want nòg die mediese professie nòg die Natalse Provinsiale Administrasie is bereid om hierdie of enige ander regering te help om die Durbanse Mediese Skool met die dwangbuis van staatsbeheer te kniehalter nie.

Hoewel die vooruitsienbare gevolge van die Regering se plan benadruk is, weet niemand waar die saak gaan eindig nie. Die beskikbaarstelling van hospitale is 'n funksie van die provinsiale owerheid, en ons verkondig slegs die vanselfsprekende as ons sê dat mediese

* Die sienswyses en vroeëre optrede van die Raad in hierdie saak is gerapporteer in hierdie Tydskrif op 30 Maart 1957 (bl. 163) en op 22 Februarie 1958 (bl. 86).

if it is the Government's intention to legislate for the transfer of the Durban Medical School to a State Department in some other way, the deputation shall reiterate to the Ministers the views of the Council thereanent.' *

In the course of the debate, Professor Gordon disclosed to the Council that at a meeting of the Faculty of Medicine held on 5 March 1958, 91 of 92 members present (there was one abstention) re-affirmed their resolve not to continue teaching activities if the Medical School were separated from the University or converted into a State-controlled institution.

He also disclosed that the Administrator-in-Executive of the Natal Provincial Council had resolved ' that the Administration will not be prepared to enter into an agreement for the joint maintenance of the Medical School with any educational authority other than the University of Natal. This is a matter of fundamental policy '.

It is now clear that much more than the University of Natal is involved. Concerned in equal measure are the Natal Provincial Administration, the South African Medical and Dental Council and the medical profession which staffs the Faculty and the teaching hospital.

Moreover, not only is the embroilment of the whole of the medical profession in Natal (if not in the rest of the country) now a reality; but we are also faced with the unedifying prospect that the Government will obtain no professional co-operation from the present establishment if it insists on carrying through its proposed plan. This means a total educational *impasse,* as neither the medical profession nor the Natal Provincial Administration will join with this or with any other Government in imposing the strait-jacket of State-control on the Durban Medical School.

Although the foreseeable consequences of the Government's undertaking have been stressed, no one can tell where the matter may yet go. The provision of hospitals is the function of the provincial authorities, and it is a glimpse of the obvious that medical students cannot be taught without hospitals (let alone without the co-operation of the medical profession).

The occasion is clearly one for statesmanship. We would therefore once again enjoin upon the Cabinet the wisdom of shedding preconceived notions and approaching this dispute in a non-partisan spirit, exhibiting an

* The views and the previous actions of the Council in this matter have been reported in this Journal on 30 March 1957 at p. 163 and on 22 February 1958 at p. 86.

studente nie opgelei kan word sonder hospitale, om nie eens van die medewerking van die mediese professie te praat nie.

Dis by uitstek 'n geleentheid waar staatsmanswysheid aan die dag gelê behoort te word. Derhalwe wil ons dit die Regering weer eens op die hart druk hoe verstandig dit sal wees om vooropgesette menings te laat vaar, om die geskil in 'n nie-partydige gees te benader, en om 'n deeglike begrip te verkry van die ernstige akademiese kwessies wat by die saak betrokke is. Sodanige verstandige leierskap aan die kant van die Regering sal die onverdeelde steun van die mediese professie in Suid-Afrika geniet.

Die Durbanse Mediese Skool ressorteer onder die bestes in die land. Die reputasie daarvan is reeds spreekwoordelik in universiteitskringe, en dis 'n toonstuk wat die wêreld met bewondering kan betrag. Die personeel het sy verpligtinge teenoor die Regering lojaal en stiptelik nagekom. Watter voorbehoude daar ook al in sekere kringe was oor die beginsel van apartheid, soos erken in die beleid van huishoudelike, interne segregasie ten opsigte van die mediese opleiding van ongegradueerdes aan die Universiteit, is die Regering se wense in hierdie verband nooit in die wiele gery nie; trouens, daar is volledige en getroue uitvoering aan daardie wense gegee. Die geskil gaan derhalwe nie oor die skending van enige ideologiese beginsel waaraan die Regering sy steun toegesê het nie. Dit is ook nie 'n politieke rassevraagstuk nie. Dit is die aard van die administratiewe beheer van 'n universiteitsinrigting wat op die spel is. Die stryd gaan om die behoud van sy akademiese kwaliteit sodat sy unieke karakter nie vernietig sal word nie. Om eensydig te volhard 'n vasberadenheid om hierdie universitêre mediese skool in 'n onderdeel van die burokratiese hiërargie van die staatsdiens te omskep, kan alleen onrus, afkeur en weerstand aan universiteitskringe en die mediese professie ontlok.

Alle moontlike pogings behoort aangewend te word om die saak tot 'n nie-politieke besprekingspeil te verhef. Dit kan gedoen word, want geen partypolitieke beginsel is in die gedrang of word bedreig nie.

Ons het reeds daarop gewys† dat die voorgestelde plan sonder betekenis is. Die uitvoering daarvan kan nie moreel geregverdig word nie. Die beweegredes bly duister. Die voordele daarvan is nog nooit verduidelik en is vir niemand begryplik nie. Waarom, dan, moet die glorierykheid van 'n groot akademiese prestasie verruil word vir iets wat geen vordering of baat kan meebring nie, en wat slegs tweedrag en 'n ramp tot gevolg kan hê?

BRITSE BEURS VIR NAGRAADSE KLINIESE STUDIE

Verlede jaar het ses Britse farmaseutiese firmas saamgespan om 'n beurs ter waarde van £600 vir nagraadse kliniese studie in die Verenigde Koninkryk gedurende 'n minimum-tydperk van 3 maande tot beskikking van 'n Suid-Afrikaanse algemene praktisyn te stel.

† Redaksioneel (1958): Hierdie Tydskrif, 22 Februarie, bl. 85.

awareness of the grave academic issues involved. Such wise leadership on the part of the government will have the undivided support of the medical profession in South Africa.

The Durban Medical School ranks with the finest in any country. It has become a byword in University circles and a show-piece for the world to look upon with admiration. Its staff have carried out their obligations to the government loyally and meticulously. Whatever reservations there may have been in certain quarters about the principle of *apartheid* recognized in the policy of domestic, internal segregation at the University in respect of under-graduate medical education, the government's wishes in this matter have never been crossed; indeed, they have been discharged fully and faithfully. The dispute therefore involves no infringement of any ideological principle to which the government adheres. There is no political racial issue here. It is the nature of the administrative control of a university institution which is at stake. The fight is to preserve its academic quality so that its unique character will not be destroyed. To press on unilaterally with a determination to convert this University Medical School into a component of the bureaucratic hierarchy of the public service will ensure only disquiet, disapproval and opposition in University circles and the medical profession.

Every effort must be made to lift the matter to a non-political level of discussion. This can be done because no party-political principle is being challenged or threatened. We have already pointed out† that the proposed undertaking is pointless. Its implementation cannot be morally justified. Its motivation remains obscure. Its advantages have never been articulated and are apparent to none. Why then must the glory of a great academic achievement be exchanged for something which cannot prosper or profit and which can only bring dissension and disaster in its wake?

BRITISH BURSARY FOR POST-GRADUATE CLINICAL STUDY

Last year six British pharmaceutical houses combined to make available, to a South African general practitioner, a bursary to the value of £600 for post-graduate clinical study in the United Kingdom, for a minimum period of 3 months.

† Editorial (1958): This Journal, 22 February, p. 85.

Die borge van die beurs was:
B.P.D. (South Africa) (Pty.) Ltd.;
British Drug Houses (South Africa) (Pty.) Ltd.;
Distillers Corporation (Biochemicals) Ltd.;
Evans Medical Supplies Ltd.;
I.C.I. South Africa (Pharmaceuticals) Ltd.;
The Crookes Laboratories Ltd.

Die beurs is deur bogenoemde organisasies beskikbaar gestel op die tydstip toe die Tweerigting-handelsveldtog tussen die Verenigde Koninkryk en Suid-Afrika van stapel gestuur is, en die bedoeling van die borge was dat die beurs as simbool moet dien van die wederkerige belange van die twee soewereine state, op 'n professionele peil, in die sfeer van farmakologiese navorsing en nagraadse kliniese opleiding. Die keurkomitee het bestaan uit: prof. G. A. Elliott; prof. F. Forman; prof. S. F. Oosthuizen; dr. H. A. Shapiro (*Voorsitter*); dr. M. Shapiro en dr. M. M. Suzman.

Op 'n voltallige vergadering van die Keurkomitee is dr. G. W. Schepers, 'n algemene praktisyn van Johannesburg, aangestel. Dr. Schepers aanvaar sy aanstelling as beurshouer in April vandeesjaar in die hoedanigheid van kliniese assistent van prof. I. Aird aan die Nagraadse Skool, Hammersmith, Londen.

By geleentheid van die vertrek van die eerste beurshouer kragtens hierdie skema is dit vir ons 'n voorreg om die volgende boodskap van prof. S. F. Oosthuizen, M.D., F.R.C.P. (Edin.), F.F.R., te publiseer:

'As Voorsitter van die Mediese Navorsingskomitee van die Raad vir Wetenskaplike en Industriële Navorsing verwelkom ek die geleentheid wat geskep word deur die beskikbaarstelling van die Britse Beurs vir Nagraadse Kliniese Studie.

In die akademiese sfeer is daar 'n aansienlike aantal toekennings vir nagraadse navorsing. Hierdie Beurs is egter 'n pioniersonderneming want, vir die eerste keer in die geskiedenis van ons land, stel dit 'n toekenning vir nagraadse kliniese studie spesiaal tot beskikking van die algemene praktisyn.

Die beurshouer wat kragtens hierdie skema aangestel is, sal nie alleen die jongste ontwikkelinge op die gebied van die diagnose en behandeling van siekte na die sfeer van algemene praktyk terugbring nie; inderdaad sal hy ook 'n belangrike gesant vir Suid-Afrika wees.

Beurse van hierdie aard open die weg tot toenemende intellektuele verkeer tussen Suid-Afrika en die Verenigde Koninkryk, en dien as aanmoediging vir die uitwisseling van mediese en wetenskaplike inligting op 'n persoonlike grondslag. Dit kan slegs tot onderlinge voordeel van albei lande strek.'

The sponsors of the bursary were:
B.P.D. (South Africa) (Pty.), Ltd.;
British Drug Houses (South Africa) (Pty.), Ltd.;
Distillers Company (Biochemicals), Ltd.;
Evans Medical Supplies Ltd.;
I.C.I. South Africa (Pharmaceuticals), Ltd.;
The Crookes Laboratories, Ltd.

The bursary was introduced by the sponsoring organizations at the time of the Two-Way Trade Campaign between the United Kingdom and South Africa. The sponsors intended the bursary to mark the reciprocal interests of the two Sovereign States, on a professional level, in the field of pharmacological research and post-graduate clinical training.

The Selection Committee consisted of: Prof. G. A. Elliott; Prof. F. Forman; Prof. S. F. Oosthuizen; Dr. H. A. Shapiro (*Chairman*); Dr. M. Shapiro and Dr. M. M. Suzman.

At a full meeting of the Selection Committee, Dr. G. W. Schepers (a general practitioner of Johannesburg) was appointed. Dr. Schepers will take up his appointment as a bursar in April this year, in the capacity of a clinical assistant to Prof. I. Aird, at the Post-Graduate School, Hammersmith, London.

We are privileged, on the occasion of the departure of the first Bursar under this scheme, to publish the following message from Prof. S. F. Oosthuizen, M.D., F.R.C.P. (Edin.), F.F.R.:

'As Chairman of the Medical Research Committee of the C.S.I.R., I welcome the opportunity provided by the establishment of the British Bursary for Post-Graduate Clinical Study.

In the academic field, there is a considerable number of awards for post-graduate research. This Bursary is, however, a pioneering undertaking because it makes available, for the first time in the history of our country, an award for post-graduate clinical study specially earmarked for a general practitioner.

The Bursar appointed under this scheme will not only bring back to the field of general practice the latest developments in the diagnosis and treatment of disease; he will also, in fact, be an important ambassador for South Africa.

Bursaries of this kind pave the way for increasing intellectual traffic between South Africa and the United Kingdom and encourage the exchange of medical and scientific information on a personal basis. This can only be mutually advantageous to both countries.'

CHLOROTIASIED

'N URINE-AFSKEIMIDDEL MET 'N DUBBELE EFFEK

Die koms van 'n nuwe mondelinge niekwikhoudende urine-afskeimiddel is ingelui deur 'n stroom van sorgvuldig gekontroleerde kliniese verslae in sulke bekende publikasies

CHLOROTHIAZIDE

A DOUBLE-DUTY DIURETIC

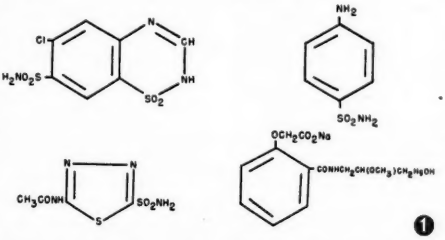
The advent of a new oral non-mercurial diuretic has been ushered in by a veritable 'rash' of carefully controlled clinical reports in such well-known publications as, *inter alia*,

soos, onder meer, die *Journal of the American Medical Association,* die *British Medical Journal, The Lancet,* die *New England Journal of Medicine* en die *Boston Medical Quarterly.*

Chemies is chlorotiasied (ook bekend as Chlotride, Saluric en Diuril) 6–chloro–7–sulfamiel—1, 2, 4–bensotiadiasien–1, 1–dioksied. Die struktuur daarvan word aangedui in Tekening 1, wat ook die struktuur van die verwante samestellings aantoon:

Hierdie samestelling word as 'n sulfonamied geklassifiseer omdat dit die SO_2NH_2 (sulfamiel)-radikaal bevat. Dit is egter nie 'n sulfanilamied-samestelling nie, want dit het 'n heeltemal verskillende chemiese struktuur en hoegenaamd *geen* bakteriebestrydende bedrywigheid nie. Chlorotiasied is 'n bensotiadiasien-derivaat, en verskil chemies van sulfanilamied.

Chlorotiasied bevat geen kwik nie.



Tek. 1. Bo, links: Chlorotiasied; *Bo, regs:* Sulfinilamied. *Onder links:* Asetasolamied; *Onder, regs:* Organiese kwikhoudende middel.

Fig. 1. Top left: Chlorothiazide; *Top right:* Sulphanilamide. *Bottom left:* Acetazolamide; *Bottom right:* Organic mercurial.

Talle uiteenlopende navorsingswerkers skyn heelhartig saam te stem dat chlorotiasied 'n kragtige effek het en in staat is om opvallende veranderings in die serum-elektrolitiese patroon teweeg te bring.

Dit is gebruik vir die behandeling van gevorderde toestande van vloeistofbehoud voortspruitende uit hartversaking, sirrose van die lewer, die nephrosis-sindroom, chroniese nierkwaal, die toksemie van swangerskap en steroïed-edeem. Die gebruik daarvan word nie aangedui in gevalle van glaukoom of epilepsie nie.

In 'n inleidingsartikel in die *British Medical Journal*[1] van 7 Desember 1957 word verklaar:

'Daar bestaan 'n dringende behoefte aan 'n doeltreffende mondelinge urine-afskeimiddel . . . 'n farmakologies oorspronklike stap was die opvolging van die waarneming dat sekere sulfonamiede as stremmers van koolsuuranhidrase optree.'

In die vordering wat in hierdie navorsingsrigting gemaak is, is chlorotiasied uiteindelik deur Novello en Sprague[2] opgebou. By die mens het die middel 'n urine-afskeidingseffek, want dit verhoog die afskeiding van chloried wat vergesel gaan van 'n byna ekwimolêre verlies van natrium. Dus, terwyl dit dieselfde effek as organiese kwikhoudende stowwe het vir sover dit die afskeiding van chloried betref, verskil dit van hulle omdat dit 'n afskeiding

1. Redaksioneel (1957): Brit. Med. J., **2,** 1355.
2. Novello, F. C. en Sprague, J. M. (1956): J. Amer. Chem. Soc., **79,** 2028.

the *Journal of the American Medical Association,* the *British Medical Journal, The Lancet,* the *New England Journal of Medicine* and the *Boston Medical Quarterly.*

Chemically, chlorothiazide (also known as Chlotride, Saluric, Diuril) is 6–chloro–7–sulphamyl–1, 2, 4–benzothiadiazine–1, 1–dioxide. Its structure is indicated in Fig. 1, which also shows the structure of related compounds.

This compound is classed as a sulphonamide because it contains the SO_2NH_2 (sulphamyl) radicle. However, it is not a sulphanilamide compound, having a completely different chemical configuration and having *no* antibacterial activity. Chlorothiazide is a benzothiadiazine derivative and differs chemically from sulphanilamide.

Chlorothiazide does not contain mercury.

The opinion of numerous and varied investigators seems in agreement on the point that chlorothiazide is potent and capable of causing striking changes in the serum electrolyte pattern.

It has been used in the treatment of advanced states of fluid retention resulting from heart failure, cirrhosis of the liver, the nephrotic syndrome, chronic renal disease, the toxaemias of pregnancy and steroid oedema. It is not indicated in glaucoma or epilepsy.

As an Editorial in the *British Medical Journal*[1] of 7 December 1957 points out:

'The need for an effective oral diuretic is urgent. . . . A pharmacologically original step was to follow up the observation that certain sulphonamides are carbonic anhydrase inhibitors.'

In the progress of this line of research, chlorothiazide was eventually synthesized by Novello and Sprague.[2] In Man, the drug is diuretic by enhancing the excretion of chloride, which is accompanied by an almost equi-molar loss of sodium. Thus, while resembling organic mercurials in chloride excretion, it differs from them in producing an excretion of sodium and chloride in physiological proportion—hence its designation as a saluretic agent.

The mode of action of chlorothiazide appears to be different from that of other known

1. Editorial (1957): Brit. Med. J., **2,** 1355.
2. Novello, F. C. and Sprague, J. M. (1956): J. Amer. Chem. Soc., **79,** 2028.

van natrium en chloried in 'n fisiologiese verhouding tot gevolg het. Dis dan ook die rede waarom dit 'n saluretica genoem word.

Die werkwyse van chlorotiasied verskil skynbaar van dié van ander bekende samestellings. Terwyl dit 'n koolsuuuranhidrase-stremmingseffek *in vitro* het, skyn dit asof die uitwerking daarvan hoofsaaklik toegespits is op die voorkoming van die her-absorpsie van natrium, chloried en water in die buise. ‚Temeer, daar is bevind dat die effek daarvan as aanvullend beskou kan word tot dié van meralluried of 'n koolsuuranhidrase-stremmer'[3] of albei, en dat dit indikatief van verskillende plekke en werkswyses is.[4]

Chlorotiasied werk vinnig (binne ongeveer 2 uur), en die effek van 'n dosis duur tussen 10 en 12 uur lank. Die maksimum-effek word binne die eerste 2 tot 6 uur waargeneem. Daar word beweer dat dit nie alleen kragtiger as ander mondelinge urine-afskeimiddels is nie, maar dat die effek daarvan ook vergelyk kan word met dié van perenterale organiese kwikhoudende middels. Temeer, daar word gerapporteer dat dit goeie urine-afskeiding tot gevolg het by pasiënte wat weerstand teen alle ander vorms van terapie bied.

'n Ander belangrike eienskap is sy vermoë om sy urine-afseidingskrag te behou selfs nadat dit herhaaldelik oor lang tydperke toegedien is. Die feit dat die pasiënt geen weerstand teen die middel ontwikkel nie, gepaard met die afwesigheid van enige betekenisvolle toksisiteit of kontra-indikasies vir die gebruik daarvan, maak chlorotiasied 'n baie waardevolle geneesmiddel, nie alleen vir die behandeling van edeem wat aan feitlik enige oorsaak te wyte is nie, maar ook vir die voorkoming en behandeling van die toksemies van swangerskap.

Hieruit vloei daar die tweede merkwaardige eienskap van die middel, nl. sy effek op hoë bloeddruk. Dit is lank reeds bekend dat die uitwerking van talle hipertensie-bestryders, veral die senuknoopversperringsmiddels, opvallend beïnvloed kan word deur die pasiënt se water- en elektrolitiese ewewig, en NaCl in besonder.

Wilkins[5] het waargeneem dat die kliniese toestande wat met vermeerderde liggaamsvloeistowwe of die behoud van natrium geassosieer is, betreklik min reaksie op hipotensie-middels

3. Laragh, John H., Heineman, Henry O. en Demartini, Felix E. (1958): J. Amer. Med. Assoc., **166,** 145.
4. Ford, R. V. (1957): J. Lab. Clin. Med., **50,** 814.
5. Wilkins, R. W. (1957): New Eng. J. Med., **257,** 1026.

compounds. While it has carbonic anhydrase inhibitor effects *in vitro,* its main action appears to be prevention of tubular reabsorption of sodium, chloride and water. 'Furthermore its effects have been found to be additive to those of meralluride or a carbonic anhydrase inhibitor'[3] or both, and are indicative of different sites and modes of action.[4]

Chlorothiazide acts rapidly (within a matter of 2 hours) and the effect of a dose lasts for some 10–12 hours. The maximum effect is in the first 2–6 hours. It has been claimed to be not only more potent than other oral diuretics, but also equal in its action to parenteral organic mercurials. Furthermore, it is reported frequently to produce a good diuresis in patients resistant to all other therapy.

Another important property is its power to retain its diuretic properties although administered repeatedly over a long period. This lack of development of drug resistance, the absence of any significant toxicity and no known contra-indications make chlorothiazide a valuable drug, not only in the treatment of oedema due to almost any cause, but also in the prevention and treatment of the toxaemias of pregnancy.

This leads to its second remarkable feature, which relates to its action in hypertension. It has long been known that the action of many antihypertensive agents, particularly the ganglionic-blocking drugs, may be markedly influenced by the patient's water and electrolyte balance, and by NaCl in particular.

Wilkins[5] has observed that clinical states associated with increased body fluid or retention of sodium are relatively unresponsive to the hypotensive agents. This is in marked contrast to conditions in which there is a depletion of body fluid and sodium, when the response to these hypotensors is markedly increased. His management of congestive heart failure in hypertensive patients logically led him to employ diuretics (in order to create water and electrolyte balance) before using a hypotensive drug. He has claimed that chlorothiazide is of outstanding benefit in such cases, particularly when used supplementary to other hypotensive drug treatment.

3. Laragh, John H., Heineman, Henry O. and Demartini, Felix E. (1958): J. Amer. Med. Assoc., **166,** 145.
4. Ford, R. V. (1957): J. Lab. Clin. Med., **50,** 814.
5. Wilkins, R. W. (1957): New Eng. J. Med., **257,** 1026.

toon. Dit staan in skerp teëstelling met toestande waar daar 'n tekort aan liggaamsvloeistowwe en natrium is; in sulke gevalle word die reaksie op hierdie hipotensie-middels opvallend verhoog. Sy behandeling van kongestiewe hartversaking by pasiënte wat aan hoë bloeddruk ly, het hom logies gebring by die aanwending van urineafskeimiddels om 'n water-en-elektrolitiese ewewig te skep) voordat hy 'n hipotensie-middel voorgeskryf het. Hy het vasgestel dat chlorotiasied in sulke gevalle van voortreflike waarde is, veral as dit die behandeling met hipotensie-middels aanvul.

Dit skyn asof chlorotiasied *per se* 'n hiptertensiebestrydingseffek het, maar op die oomblik word daar allerweë gemeen dat dit sy behoorlike rol as 'n hipotensie-middel speel alleen as dit saam met ander middels gebruik word. Dit maak die behandeling van hoë bloeddruk heelwat buigbaarder, en verbeter kontrole. Terselfdertyd stel dit die geneesheer in staat om die onaangename newe-effekte van sommige hipotensie-middels te verminder, of heeltemal uit te skakel.

Dit is derhalwe nie verrassend nie dat chlorotiasied houdings-hipotensie oordryf. Dit is iets wat in gedagte gehou moet word wanneer die middel toegedien word saam met senuknoopversperringsmiddels om houdings-induiestorting te voorkom.

Navorsingswerkers oor die algemeen rapporteer dat wanneer chlorotiasied gevoeg word by die middels waarmee nie-edeemagtige hoë-bloeddruk-pasiënte behandel word, dit noodsaaklik is om die dosis senuknoopversperringsmiddels dadelik met soveel soos 50% te verminder, en om dit daarna te wysig om maksimum-effek te verkry. Die vermindering van die senuknoopversperringsmiddels kom gemiddeld op sowat 40% te staan.

Hollander en Wilkins[6] benadruk die doeltreffendheid van chlorotiasied by die beheer van bloeddruk in ‚hoogs weerstandskragtige gevalle van hipertensie, veral dié wat weerstand teen splanchinektomie bied.'

Chlorotiasied se unieke hipotensie- en urine-afskeidingseienskappe word benadruk deur Wilkins[5] wat hom soos volg uitlaat:

‚Dit het geen hopitensiewe of opvallende urineafskeidingseffek by kontrole-pasiënte met normale bloeddruk nie; dit het geen hipotensie-effek by normaal-tensiewe pasiënte wat aan kongestiewe versaking ly nie, maar by hulle het dit wel 'n opvallende urine-afskeidingsuitwerking; dit is hipotensief by sowel gekompenseerde as gedekompenseerde hipertensie-pasiënte (in eersgenoemde geval, sonder kongestiewe hartversaking, het dit geen opvallende urine-afskeidingseffek nie, maar in laasgenoemde geval, met kongestiewe versaking, het dit 'n opmerklike urine-afskeidingsuitwerking) . . . .' Daar word dus gerapporteer dat die middel 'n nie-urine-afskeidingseffek het by nie-edeemagtige pasiënte en 'n nie-hipotensie-effek by nie-hipertensie-pasiënte.

Uit die massa sorgvuldige navorsingswerk waaroor daar reeds verslag gedoen is, blyk dit betreklik duidelik dat chlorotiasied definitief beskryf kan word as 'n middel met 'n tweeledige effek by die behandeling van sowel hoë bloeddruk as edeem. Maar hierdie kragtige terapeutiese aanwins moet met versigtigheid gebruik word. Uit die standpunt van

6. Hollander, W. en Wilkins, R. W. (1957): Boston Med. Quart., **8**, 69.

Chlorothiazide appears to be antihypertensive *per se*, but at present there seems general agreement that its proper role as a hypotensive agent is in combination with other drugs. This introduces great flexibility and improved control into the management of hypertension and, at the same time, permits reduction or elimination of the undesirable side effects of some hypotensive agents.

It is also not surprising that chlorothiazide will exaggerate postural hypotension. This must be borne in mind when the drug is given in conjunction with ganglion-blocking agents, in order to prevent postural collapse.

Investigators generally report that when chlorothiazide is added to the regimen of non-oedematous hypertensives it is necessary immediately to reduce the dosage of the ganglionic blocking agent by as much as 50% and then adjust to obtain optimal effects (averaging about a 40% reduction in the ganglionic agent).

Hollander and Wilkins[6] have stressed the efficacy of chlorothiazide in controlling the blood pressure of 'highly resistant cases of hypertension, especially those resistant to splanchnicectomy'.

Chlorothiazide's unique hypotensive and diuretic properties are emphasized by Wilkins[5] who states:

'It is not hypotensive (or markedly diuretic) in normotensive control subjects; it is not hypotensive in normotensive patients with congestive failure, in whom it is markedly diuretic; it is hypotensive in both compensated and decompensated hypertensive patients (in the former without congestive heart failure, it is not markedly diuretic, whereas in the latter in congestive failure, it is markedly diuretic). . . .'

Thus the drug is reported to be non-diuretic in non-oedematous subjects and non-hypotensive in non-hypertensive patients.

It seems fairly clear, from the mass of careful investigation which has been reported, that chlorothiazide can definitely be described as a dual purpose drug in the management both of hypertension and of oedema. But this potent therapeutic acquisition should be used with due respect. From the standpoint of economy and ease of administration its position may be pre-eminent. However, during prolonged administration of the drug at maximum dosage levels, it would be wise to do periodic determinations of the blood chemistry, since it has such potent effects on the serum electro-

6. Hollander, W. and Wilkins, R. W. (1957): Boston Med. Quart., **8**, 69.

besuiniging en maklike toediening beklee dit 'n uiters belangrike posisie. Tydens die langdurige toediening van maksimum-dosisse van die middel sal dit egter verstandig wees om die bloedskeikunde van tyd tot tyd te ondersoek, aangesien dit so 'n kragtige effek op die serum-elektroliete het en aanleiding tot kaliumuitputting kan gee (wat egter maklik gekontroleer kan word deur 'n kaliumchloried-toevoegsel).[7]

In gevalle van hoë bloeddruk is kleiner dosisse van die middel 1 tot 3 keer per dag gebruik, maar in gevalle van edeem sal toediening op 'n hoër peil gedurende 4 agtereenvolgende dae, en staking gedurende die daaropvolgende 3 dae gewoonlik die pasiënt in stand hou, en tegelykertyd ernstige kaliumuitputting voorkom deur die herstel van die elektrolitiese ewewig moontlik te maak.

Dit is moontlik dat die presiese wyse waarop en die plek waar chlorotiasied te werk gaan nog nie vasgestel is nie. Die gebruik daarvan sal ongetwyfeld hernude belangstelling aanwakker in die fisiologie van die nierbuisies. Afgesien van die verdere lig wat die middel op die funksies van die nier kan werp, skyn dit asof die kliniese belangrikheid daarvan reeds op 'n hegte grondslag berus, en dit moet voortaan ongetwyfeld erken word as 'n belangrike en doeltreffende wapen by die bestryding van edeem en hipertensie.

7. Bayliss, R. I. S., Marrack, D., Pirkis, J., Rees, J. R. en Zilva, J. F. (1958): Lancet, **1**, 120.

lytes and may lead to potassium depletion (readily controlled by a potassium chloride supplement).[7]

In hypertension the drug has been administered in smaller doses 1 to 3 times daily, but in oedematous states administration at higher levels for 4 consecutive days and discontinuation on the 3 following days usually maintains the patient but avoids serious potassium depletion by allowing re-establishment of electrolyte balance.

It is probable that the exact mode and site of action of chlorothiazide is not yet precisely known. Its use will undoubtedly stimulate renewed interest in the physiology of the kidney tubules. Nevertheless, whatever further light the drug may throw on kidney function, its clinical importance seems to have been established and it must henceforth undoubtedly be recognized as a valuable drug in the management of oedema and hypertension.

7. Bayliss, R. I. S., Marrack, D., Pirkis, J., Rees, J. R. and Zilva, J. F. (1958): Lancet, **1**, 120.

ACUTE POLIOMYELITIS*

A STUDY OF THE CLINICAL MANIFESTATIONS OF FIFTY CASES

SEEN AT THE CHILDREN'S HOSPITAL, JOHANNESBURG, DURING THE 1948 EPIDEMIC

WITH SPECIAL REFERENCE TO THE MANAGEMENT IN THE ACUTE PHASE

J. L. BRAUDO, M.B., B.CH., M.R.C.P. (EDIN.)
Johannesburg

This survey records the clinical manifestations in 50 children afflicted with acute anterior poliomyelitis during the 1947–8 Johannesburg epidemic. The diagnosis was made on a clinical basis in all instances. All the cases were seen by the author through the acute stage and again two years later.

The clinical features of poliomyelitis have altered but little over the past 10 years. It was therefore considered appropriate to present the clinical aspects, the handling in the acute phase, and the prognosis of these 50 cases. Furthermore, certain aspects of the clinical features in this series were compared with those seen in epidemics in other parts of the world, viz. America, Malta and South Australia. The report will conclude with a separate section on the differential diagnosis of acute poliomyelitis.

* The References will be published at the end of the concluding article in this series.

I. ETIOLOGY, EPIDEMIOLOGY, HISTORY AND SEASONAL INCIDENCE OF ACUTE POLIOMYELITIS IN JOHANNESBURG

ETIOLOGY

The experimental production of poliomyelitis was first accomplished in 1909.[1] The infecting agent belongs to the group of filterable viruses.

There are 3 main groups of poliomyelitis viruses: type I (e.g. Brunhilde), type II (e.g. Lansing) and type III (e.g. Leon). Analyses of epidemics have shown that type I virus has been responsible for most major epidemics, type III for an occasional one, but no major

epidemic has yet been attributed to type II virus. However, antibodies to the Lansing strains have commonly been found in all parts of the world where endemic poliomyelitis exists.[2]

In this epidemic the poliomyelitis virus was isolated from numerous cases. The Brunhilde type I virus was recovered from most of the specimens examined.[3]

EPIDEMIOLOGY

The study of the mode of transmission of epidemic poliomyelitis has been well clarified by the observations of Wickman.[4] He drew special attention to its contagious character, and his description of the so-called abortive and meningitic forms threw an entirely new light on the obscure question of dissemination. Until then attention was only focussed on the paralytic cases and the possibility that epidemic poliomyelitis could occur without paralysis was not entertained. Since poliomyelitis occurs in epidemics, it must be an infectious disease. Furthermore, the infective agent finds a considerable degree of resistance naturally present in any community. This is proven by the strong tendency of the frankly paralytic types of the disease to appear as isolated cases in a given family or household. However, the occurrence of dual and even multiple infection of the non-paralytic type in one household is not uncommon. In this series involving 14 cases, there were 6 instances of multiple infection with various forms of the disease. In the 1947–8 Johannesburg epidemic there were some 30 instances of multiple infection involving 68 cases out of a total of 623 notified cases.[5] These figures show that direct contact played an important role in the spread of the disease.

HISTORY OF POLIOMYELITIS IN JOHANNESBURG

Poliomyelitis has been endemic in Johannesburg for many years but no widespread outbreak occurred until towards the end of World War I in 1918 when the first epidemic took place. There were 145 cases of which only 6 (4%) occurred in the non-European population. During the following 20 years only 64 cases were notified. From 1939–44, 85 cases were reported and then at the end of World War II followed the 1944–5 epidemic with 201 cases of which 27 (17%) were in non-Europeans. Between 1945 and 1947 only 55 cases were reported. The biggest epidemic occurred in 1947–8 when 721 cases were reported, of which 118 (16%) were in non-Europeans.[5]

From these figures it appears that there has been an increase in the notified incidence of poliomyelitis in the population as a whole. It is possible that a large number of unknown cases did occur which were not included in the public health records. When considering the significance of these figures it must be noted that of a total population of 839,154 in Johannesburg in 1948, 343,192 (about 40%) were Europeans, and consequently the case incidence was 177 per 100,000 in the European population and only 26 per 100,000 in the non-European.[5] The inferior nutrition and unsanitary living conditions of the non-European probably contributed to his comparative resistance to the poliomyelitis virus. This is supported by the fact that there was a much higher case incidence in the well-nourished and well-housed European section of the population than in the poorly nourished, poorly housed section. These findings are supported by Sabin[6] in the North Eastern and Southern parts of the U.S.A. He states:

> '. . . poverty and the poor hygienic conditions that go with it might explain the lower attack rates among the Negroes in the North East and among both the Whites and Negroes in the Southern areas.'

SEASONAL INCIDENCE OF POLIOMYELITIS

Poliomyelitis has always been a disease of the late summer and early autumn in all parts of the world.

Five hundred and fifteen cases (80%) of a total of 600 occurred between November 1947 and April 1948, with the greatest incidence in the months of February, March and April.[5]

II. CLINICAL TYPES, INCUBATION PERIOD, PRODROMAL PATTERNS AND PATHOGENESIS OF ACUTE POLIOMYELITIS

CLINICAL TYPES

The first recorded observations of the clinical phase of poliomyelitis was probably made by Underwood.[7] He recognized that children might suddenly become paralysed after a brief illness. Badham[8] in 1835 presented a much more definite description. He reported 4 remarkable cases of paralysis suddenly induced in the extremities, occurring in children without any apparent cerebral or cerebrospinal lesion. These children also had pain in the affected extremities, relieved by warmth. The

children lived in the same community and this outbreak might be regarded as having been a minor epidemic.

In 1840 the first systematic study of the clinical picture was made by Heine, a German orthopaedic surgeon.[9] He was the first clinician to realize that this peculiar paralytic disease was always preceded by an acute febrile period. He also demonstrated that there was a febrile reaction which might be so mild as not to arouse any anxiety in the minds of the parents, and pointed out the great contrast between their reaction during the acute stage and later, when they suddenly noticed a paralysed limb on the morning following the acute symptoms. Heine[9] recorded that pain in the extremities, fever, irritability and convulsions, preceded paralysis in most cases.

The literature is barren of any discussion about poliomyelitis until Strümpell[10] in 1884 described the encephalitic, neuritic and poliomyelitic forms of the disease. He also suggested its infectious nature. The next important contribution was that of Medin,[11] who published details of the large Swedish epidemic. He gave an excellent description of the acute phase and suggested that it was an acute systemic infection as well as a paralytic one. Caverly in 1896[12] first described rigidity of the spinal muscles in 6 cases of poliomyelitis without paralytic manifestations.

Then followed Wickman's classic monograph in 1913.[4] He described 8 forms of the disease:

i. The spinal poliomyelitic form.
ii. The form resembling Landry's ascending paralysis.
iii. The bulbar or pontine form.
iv. The encephalitic variety.
v. The ataxic type.
vi. The polyneuritic type.
vii. The meningitic form.
viii. The abortive variety.

Wickman noted all the clinical features seen in the more recent epidemics, and recorded neck and spinal stiffness, pain of various types and all the clinical varieties of the disease, while his remarks on therapy were almost the same as those of the modern physician. Ever since he stressed the abortive type of the disease, viz. those cases which developed neither paralysis nor symptoms of central nervous system invasion, observers all over the world have recognized its importance. The exact number of cases belonging to this form of the disease varies greatly, but it represents at least 50% or more of the total incidence of the disease.

Several examples of the febrile disease alone, or in combination with an upper respiratory infection, gastro-enteritis or 'influenza', appeared in the families of the cases of poliomyelitis described in this report. Paul and Trask[13] on the basis of house surveys carried out in 1931 and in 1932, estimated the ratio of abortive to frank cases at about 9 : 1. Recent investigations have shown that the ratio of abortive to paralytic cases had been underestimated. Sweetman[14] stated they might number 20 : 1 or even several hundred to one as estimated in a recent epidemic in New Zealand, whereas Turner *et al.*[15] estimated that there were probably 1,000 sub-clinical poliomyelitis infections with the Lansing strain for every clinical one.

THE CLINICAL PATTERN

INCUBATION PERIOD

In the experimental disease in monkeys Flexner and Lewis[16] found that the time between inoculation with the poliomyelitis virus and the onset of paralysis varied from 3 to 33 days, with an average of 8 to 9 days. Howe and Bodian,[17] by oral and intra-gastric administration of virus-containing human stools, produced paralytic poliomyelitis in 2 chimpanzees after incubation periods of 12 and 28 days respectively. The average incubation period in man varied from 3–35 days, the commonest being 7–12 days. In the 1947–8 Southern Australian epidemic the incubation period determined clinically in 25 cases varied from 6–35 days, with an average of 11 days.[18] In this series of 50 cases the incubation period ranged from 4–14 days. Two cases occurred 4 and 5 days respectively after tonsillectomy. One appeared 8 days after direct contact and another 14 days after admission to hospital for an osteitis of the right humerus.

The incubation period in human poliomyelitis is not subject to exact analysis because of gaps in our knowledge of the epidemiology of the disease. There is both difficulty and disagreement regarding the exact points at which the incubation period begins and ends. Incubation periods are estimated clinically from contact with known cases as well as from those occurring after tonsillectomy. In experimental poliomyelitis, however, the exact incubation period can be determined, since the virus is introduced into the animal at a known time.

PRODROMAL STATE

Following the incubation period prodromata occur in most cases. Draper[19] emphasized the occurrence of prodromal phenomena which

cleared up entirely, so that for a day or two the child appeared perfectly well. Nevertheless, one or two days later, and even without warning, the disease commenced and the child became paralysed. He called this series the 'dromedary group', because of the 2 'humps' of symptoms. In the second series, a so-called 'straggling group', this stage of comparative well-being was not present, but there was a sustained period of indisposition of varying intensity leading to the invasion of the central nervous system. In the third, or 'sudden group', all the initial signs pointed to the invasion of the central nervous system.

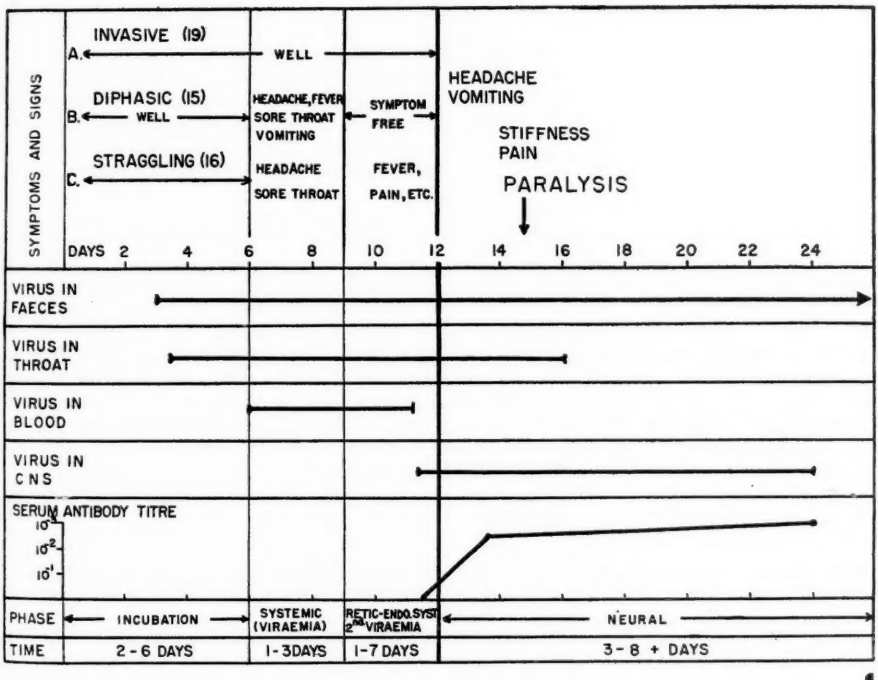
The foregoing clinical pattern of the disease has recently been emphasized by Horstman,[20] who substituted the term 'diphasic' for 'dromedary'. She found that the first phase of the illness lasted for 1–2 days, and was then followed by a period of 1–7 days before the actual invasion of the central nervous system began (Fig. 1).

In this series the clinical pattern followed very closely that described by Draper[19] and Horstman.[19] The diphasic or dromedary variety occurred in 14 cases, the straggling type in 16 cases and the invasive or sudden type in 20 cases (Table 1).

The duration of the first phase of the diphasic form varied from 1–3 days with an average of 1.7 days, while the interval between the 2 phases ranged from 0.5–6 days, with an average of 2.3 days.

In the straggling variety the first phase lasted from 1–7 days with an average of 4 days. These figures were very similar to those of Horstman,[20] who stated that the diphasic course was common in young children, and that the time between the onset of the first phase and the development of the disease proper was shorter in children than in adults.

Previous attempts to explain the clinical pattern of the disease on a pathological basis accepted the principle of strict neurotropism of the virus. Draper[19] suggested that the disease had an initial systemic phase due to blood stream invasion, but could not prove it. It was not until recently that the hypothesis of strict neurotropism of the poliomyelitis virus was scientifically challenged.



1

Fig. 1. Clinical patterns and phases of poliomyelitis. [*After* Bodian, D. (1952): *Pathogenesis of Poliomyelitis*, Amer. J. Pub. Hlth., **42**, 1388.]

TABLE 1: PRODROMAL PATTERNS OF ACUTE POLIOMYELITIS

	DISPHASIC OR DROMEDARY PATTERN (15 Cases)			
	Incubation Period	*First Phase*	*Interval*	*Second Phase*
Symptoms	Asymptomatic	Fever, malaise, headache, sore-throat	Asymptomatic	
Time	Probably 5—15 days	1—3 days 1·7 days (average)	0·5—6 days 2·3 days (average)	1—8 days
	7—14 days	1—2 days	1—7 days	3—8 days
	Horstman (1949)	Horstman (1949)	Horstman (1949)	Horstman (1949)
	STRAGGLING PATTERN (16 Cases)			
	Incubation Period	*First Phase*	*Interval*	*Second Phase*
Symptoms	Asymptomatic	Fever, headache, malaise, sore-throat	No interval	Merges with second phase
Time	5—15 days	1—7 days 4 days (average)		
	INVASIVE PATTERN (19 Cases)			
	Incubation Period			
Symptoms	Asymptomatic	First and second phase combined.		
Time	5—15 days			

Firstly, Enders *et al.*[21] succeeded in cultivating the Lansing strain of the poliomyelitis virus in various human embryonic tissues. This work was confirmed by Ledinko *et al.*,[22] who were able to initiate multiplication of the poliomyelitis viruses in tissue cultures of monkey testes. Although sporadic isolation of the virus from the blood and extra-neural tissues[23] of patients afflicted with poliomyelitis, e.g. lymph nodes[24] and ileum[25] have been reported, the role of viraemia in poliomyelitis has recently come to the fore again.

Bodian[26] and Horstman,[27] working with experimental animals demonstrated the regular presence of viraemia in the incubation period after simple virus feeding in Cynomolgus monkeys and in chimpanzees. Furthermore, the virus has also been isolated from the blood of an early abortive case of the disease. Bodian,[26] taking these findings into consideration, postulated 3 phases in the dissemination of the virus in 'natural' human infection after simple virus feedings in chimpanzees and Cynomolgus monkeys (Fig. 1). The portal of entry and exit is the alimentary mucosa and the principal method of spread is person-to-person contact, but it is not yet clear whether oropharyngeal or faecal material is the important vehicle.

From the bowel the virus enters the blood stream directly or possibly via the regional lymph glands and then the reticulo-endothelial system (from which it may also emerge into the blood stream). The latter phases represent the vascular one.

The neural phase then ensues. The virus leaves the vascular system and may enter the central nervous system via the area postrema in the medulla oblongata, before its dissemination throughout specific areas of the central

nervous system, especially the motor nuclei in the brain stem and the spinal cord. In some instances it is conceivable that the virus enters the central nervous system directly from the alimentary musoca via regional nerves and ganglia. Meenan[28] regards it as legitimate to extend Bodian's hypothesis and adduce evidence from other virus diseases.

'The virus', he says, 'travels from the site of entry to regional lymph nodes where it multiplies. This is followed by invasion of the blood stream via which other lymphoid tissues, e.g. spleen, bone-marrow, lymph glands, etc. are infected. After a further period of growth a secondary viraemia occurs during which the virus travels to those cells which, when attacked, give the clinical picture of the disease'.

With these data the explanation of the 3 clinical patterns of the disease would be as follows:

The first hump of the dromedary type would correspond to the invasion of regional lymph glands and initial viraemia. Then follows the asymptomatic period of virus multiplication in the reticulo-endothelial system. The second hump is due to the secondary viraemia leading to invasion of the central nervous system. On the other hand, the straggling type is probably due to the same factors producing the first hump of the dromedary variety, but the reticulo-endothelial phase is symptomatic. In the invasive type, however, the initial invasion of lymphatic glands, blood and reticulo-endothelial system is asymptomatic, and the disease expresses itself clinically with the emergence of the second viraemic episode and central nervous system disorganization.

This concept of pathogenesis can likewise explain the 3 clinical varieties of the disease:

i. The most common form, the abortive attack in which invasion and viraemia occur, but antibody response is sufficiently adequate to prevent the further progress of the disease.

ii. The non-paralytic variety in which the central nervous system is invaded, but paralysis does not ensue.

iii. The paralytic variety in which, for reasons as yet inadequately understood, invasion of the central nervous system is accompanied by sufficient disorganization to result in paralysis and even death.

The possible effects of virulent strains, poor or very slow antibody responses, seasonal factors, physical activity, pregnancy, trauma, endocrine factors, e.g. cortisone, and concurrent infection with other agents, are slowly being evaluated.

SUMMARY

The history of epidemic poliomyelitis in Johannesburg since 1918 is described.

The striking disparity between the incidence of clinical poliomyelitis in the non-European (26 per 100,000 population) and the European community (177 per 100,000) is noted. The unhygienic living conditions of the Bantu community probably account for the low incidence of clinical poliomyelitis.

The Brunhilde type I poliomyelitis virus was recovered from most of the stools examined in the 1948 epidemic.

There were 6 instances of multiple infection in a single family in this series.

Most of the cases of poliomyelitis in the 1948 Johannesburg epidemic occurred towards the end of summer and early autumn as in other countries.

The clinical forms of the disease are divided into

(*a*) Non-paralytic (17 cases);

(*b*) Paralytic, of which there were 3 varieties:

i. Spinal (19 cases);

ii. Bulbar (8 cases);

iii. Bulbo-spinal (6 cases).

The incubation period ranges from 4–14 days in cases where it could be ascertained. In the experimental form of the disease the period between inoculation and onset of paralysis varies from 3–33 days (average 8–9 days).

The three patterns of the prodromal state, viz. *diphasic, straggling* and *invasive,* were prominent in this series. They were present in 15, 16 and 19 cases respectively.

The pathogenesis of poliomyelitis in the light of modern work is described. The portal of entry is the alimentary tract. The virus then reaches the reticulo-endothelial system and the blood stream and ultimately the central nervous system.

OPSOMMING

Die geskiedenis van epidemiese poliomiëlitis in Johannesburg sedert 1918 word in oënskou geneem.

Die opvallende verskil tussen die voorkoms van kliniese poliomiëlitis onder nie-blankes (26 per 100,000 pasiënte) en onder die blanke bevolking (177 per 100,000) word beklemtoon. Die lae voorkoms van kliniese poliomiëlitis onder die Bantoegemeenskap moet waarskynlik toegeskryf word aan hul onhigiëniese lewenstoestande.

Poliomiëlitisvirus van die Brunhilde-tipe I is herwin uit die meerderheid van die ontlastingsmonsters wat tydens die 1948-epidemie ondersoek is.

In hierdie reeks was daar 6 gevalle van veelvoudige infeksie in 'n enkele familie.

Tydens die 1948-epidemie in Johannesburg het die meeste gevalle van poliomiëlitis teen die einde van die somer en gedurende die vroeë herfsmaande voorgekom—net soos in ander lande.

Die kliniese vorms van die siekte is soos volg ingedeel:

(*a*) Nie-paralities (17 gevalle);
(*b*) Paralities, waarvan daar weer drie soorte is:
i. Ruggraatpoliomiëlitis (19 gevalle);
ii. Bulbêre poliomiëlitis (8 gevalle);
iii. Bulbêre-ruggraatpoliomiëlitis (6 gevalle).

Die ontkiemingstydperk het gewissel van 4–14 dae in die gevalle waar dit vasgestel kon word. Wat die eksperimentele vorm van die siekte betref, wissel die tydperk tussen inenting en die aanvang van verlamming van 3 tot 33 dae (gemiddelde 8–9 dae).

Die kliniese vorms van die siekte is soos volg stand, nl. *tweefasig, versprei* en *invallend,* was opmerklik in hierdie reeks. Hulle was waarneembaar onderskeidelik in 15, 16 en 19 gevalle.

Die patogenese van poliomiëlitis in die lig van moderne navorsingswerk word beskryf. Die poort van binnekoms is die spysverteringskanaal. Die virus bereik vervolgens die reticulo-endothelium-stelsel en die bloedstroom, en uiteindelik ook die sentrale senuweestelsel.

(To be continued)

THE SURGERY OF THE GALL BLADDER AND THE BILE DUCTS*

A. LEE MCGREGOR, M.CH. (EDIN.), F.R.C.S. (ENG.)†

Honorary Research Associate, Department of Surgery, University of Witwatersrand, Johannesburg

The patient comes to the theatre with a Levin tube in the stomach.

THE APPROACH

Whether the incision be vertical, transverse or oblique is a matter of choice. The oblique is the best, whatever the patient's build. Vertical abdominal incisions are being used less and less. They cut across the fibres of the rectus sheath, which runs transversely, and weaken the abdominal wall. They are followed by a greater percentage of scar herniae than are transverse or oblique incisions.

In sthenic persons the incision is made just below the costal margin; in hyposthenic cases it may be made an inch lower. It should extend several inches to the left of the midline. Skin towels are applied. The skin incision extends well to the right of the ninth costal cartilage, i.e. outer border of rectus (Fig. 1).

This is because many candidates for this operation are fat, hence wide exposure is necessary. The incision through the rectus sheath begins at the left inner border of the left rectus, cuts through the linea alba and the whole extent of the right anterior rectus sheath. It stops at its outer border. The only muscle divided is the right rectus and, if required, part or all of the left one.

The right rectus muscle is cut through boldly in its whole width. This enables all bleeders to be seen and secured with artery forceps. The measure of a skilled assistant is that he will handle these forceps so gently that none of them is torn off. The deeper tissues are never divided lateral to the outer edge of the rectus sheath, as the ninth and tenth intercostal nerves would be cut. The eighth nerve can often be seen and protected where it lies on the posterior rectus sheath. This sheath is cut across, opening the peritoneum.

Exploration is then carried out. Stomach, duodenum, liver and other abdominal contents are examined. Occasionally unsuspected pathology may be encountered and, if necessary, it may be dealt with at the same operation. The

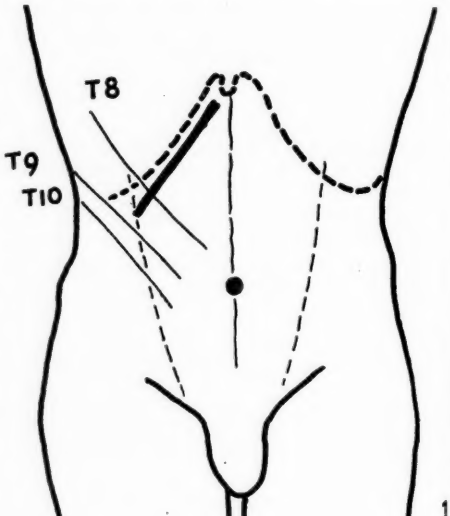


Fig. 1. Kocher's incision. The standard approach to the gall bladder.

* This is the second article in the series *Operations of Election.—Editor.*

†Consulting Surgeon, Johannesburg General Hospital.

gall bladder, the bile ducts and the pancreas are palpated.

A word of warning is necessary about palpating stones in the duct. They may be displaced into the liver and give difficulty in their removal. In one such case it was not possible to find the stone which had been displaced proximally by palpation. Some months later it gave rise to symptoms which necessitated further operation. Some stones in the bile duct are readily broken. If they are broken in handling, it is difficult to dislodge all the fragments, and any portion not removed may form a nucleus for later stone formation.

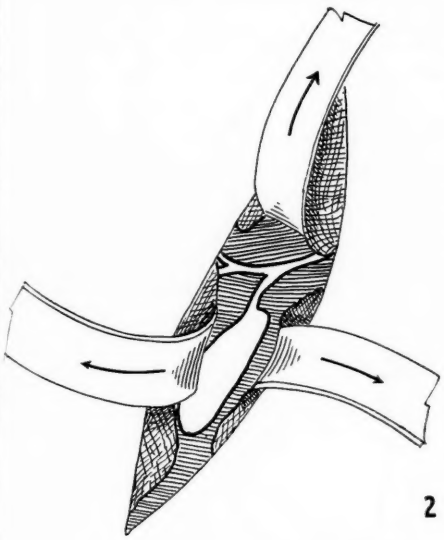


Fig. 2. The crux of the operation—the placing of the swabs.

Placing the Swabs. It makes all the difference between a good and a bad gall bladder operation if the swabs are properly disposed, to begin with.

Should there be adhesions between the gall bladder and the omentum or the colon, they are first liberated. When the bladder has been thus freed, No. 1 swab is inserted. It is a large pack, 15″ x 36″. The Sister and the opposite assistant hold the swab fully opened and the one border is placed between the liver above and the intestine below, and the pack then paid in with a 14″ non-toothed forceps in such a fashion that the hepatic flexure does not creep into the area above the swab. A broad Deaver-type retractor holds this swab distally.

No. 2 swab is placed medially, holding the duodenum away from the operation area with a No. 2 Deaver retractor. No. 3 swab is placed on the liver from the upper edge of the wound and held there with a No. 1 Deaver. This retractor has a gentle curve and is thus not likely to be forced into liver substance (Fig. 2).

Removal of the Gall Bladder: Step 1. The medial part of the gall bladder (Hartman's pouch) is grasped with ring forceps and pulled distally, exposing peritoneum over and between the cystic duct and the liver. The peritoneum is cut vertically with a knife, care being taken that only the serous membrane is divided. The inner part of the peritoneum is then pushed gently medially with a Lahey swab on a pair of Spencer Wells forceps. This swab is the size of a pea. By gentle swab dissection the cystic artery is exposed. An aneurysm needle carrying a No. 40 linen ligature is passed round the artery as much medially as possible. This ligature is tied and cut. The ring forceps is removed from Hartman's pouch and placed on the distal end of the gall bladder.

Through a 3 inch needle attached to a 20 c.c. syringe saline is now injected under the serous coat of the gall bladder, between it and the liver above and the gall bladder and the peritoneum below. The serosa balloons out because of the saline and forms a plane of cleavage. It is cut through half an inch from liver. This fringe is used later to peritonealize the gall bladder bed. The gall bladder is now dissected from its bed, using blunt-ended scissors. Any strands of tissue between the liver and the gall bladder are secured and tied. They may be veins or accessory bile ducts. When the inner end of the gall bladder is reached, the anatomy of the area is carefully surveyed and nothing is divided unless the surgeon is quite sure of what he is doing. The cystic duct is traced distally to its junction with the common hepatic duct. The hepatic artery may overlie or be to the right of the bile duct.

The triangle between the cystic duct below, the hepatic duct medially and the porta hepatis above varies greatly in its extent and its contents (Fig. 3). This is the danger zone.

With good lighting, properly disposed swabs and the mobilized gall bladder as a handle, the surgeon can be absolutely certain of what he is doing. Here again the Lahey swab is an invaluable 'pusher'. Should a fairly thick ($\frac{1}{8}$ inch or more) structure be found passing to the cystic duct from the liver,

it may be the right hepatic duct (Fig. 4). This must be protected. The abnormality is fortunately rare.

Although there is free anastomosis of blood between all parts of the liver, there is very little anastomosis of bile between the right and the left lobes, so that both the hepatic ducts are necessary to the patient.

The cystic artery, previously ligatured, may now be cut. The cystic duct is palpated and, when free of stone or grit, it is doubly clamped half an inch from the main bile ducts, and cut across. The proximal end is transfixed with a fine half-circle needle carrying a strand of No. 40 linen thread, and is ligatured. The ablated gall bladder is now cut open and inspected. The gall bladder is often tense and stones may not be felt in it, though they are present.

Indications for Opening the Common Bile Duct. The common duct should be opened in the following circumstances:

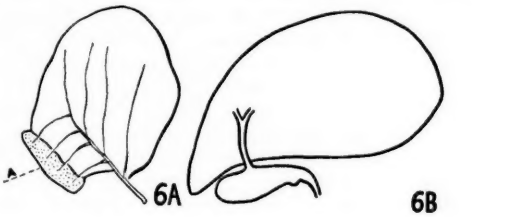
Fig. 3. X shows the danger zone.
Fig. 4. The right hepatic duct enters the cystic duct—a truly diabolical trap for the surgeon.
Fig. 5. The sessile gall bladder.

1. If it contains stones.
2. If it is dilated.
3. If the gall bladder contains small stones or gravel.
4. If the patient is jaundiced or has suffered from jaundice. This indication does not apply to the mild jaundice which so often accompanies acute cholecystitis.
5. If the gall bladder is sessile, i.e. has a large opening into the bile duct (Fig. 5).
6. If the gall bladder is fibrosed or shrunken.
7. If in doubt.

Should there be no indication for exploring the common duct, the peritoneum over the right free border of the lesser omentum is closed with a running suture of 000 plain catgut on a curved, round-bodied atraumatic needle. Care is taken to see that the cystic duct stump is buried. Bile does not clot; therefore the cystic duct stump heals from without and not from within and this process is facilitated when it abuts on peritoneum or other tissue. The suture, having reformed the serous covering of the lesser omental free border, continues on to peritonealize the gall bladder bed by picking up the peritoneal cuffs which were formed for that purpose.

Unless the appendix presents in the wound, it is not removed since it cannot, as a rule, be brought into the operative field without weakening the wound by enlarging it or using unjustifiable traction and retraction. Should there be an indication for appendicectomy, then the exposure should be through a vertical and not a subcostal incision.

Drainage. Drainage is mandatory in every cholecystectomy or operation on the bile ducts. There are excellent reasons for this. Accessory



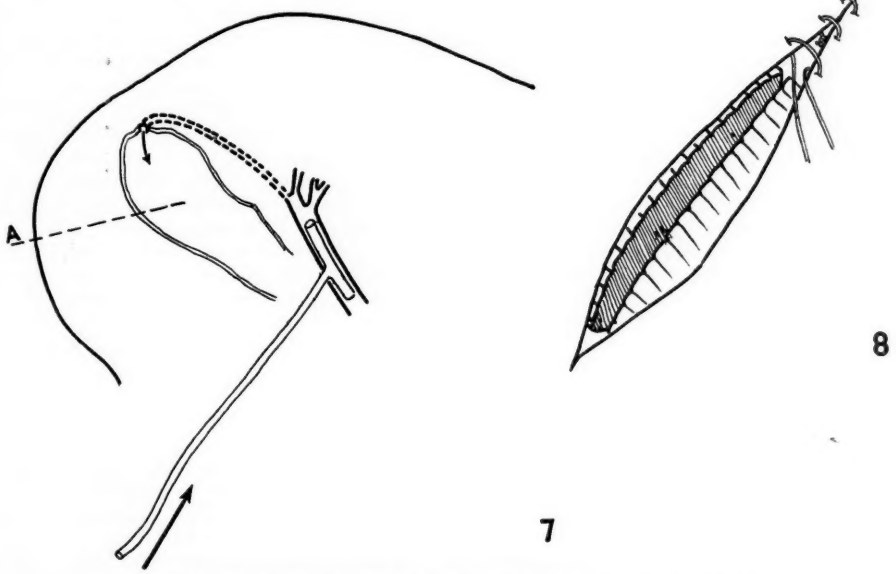


Fig. 6 A. Vestigial bile canaliculi entering gall bladder directly. *A*=Gall bladder.

Fig. 6 B. A large vestigial bile canaliculus enters the gall bladder directly.

Fig. 7. Saline injected into the tube spurts out from the periphery of the gall bladder bed through a vestigial bile canaliculus opening into the gall bladder. A=Gall bladder bed.

Fig. 8. Interrupted sutures approximating linea alba.

bile ducts occur not infrequently. During the development of the gall bladder a section of liver becomes detached from the liver anlage by deep clefts. Thus many bile channels connect the two structures (Figs. 6A, 6B).

Ultimately these are obliterated by fibrosis. Their blind ends can be found in the gall bladder bed. Coller has advised using them as a means of bile drainage in jaundice due to otherwise irremediable obstruction at the porta hepatis. One or more of these primitive bile channels may persist and will be cut across and probably not noticed, during cholecystectomy.

Only recently the following strange experience befell the writer. A chronically inflamed gall bladder with stones was removed and stones cleared from the bile duct. After the insertion of a T-tube, it was irrigated to see that the T-tube bile duct junction was watertight. The saline used spurted out from the extreme right edge of the liver, where it formed the periphery of the gall bladder bed (Fig. 7).

The duct opening was seen and ligated. Very often such openings are not seen. Bile peritonitis is a grave condition which is often fatal.

A Penrose soft rubber drain with a gauze wick in its lumen is inserted through a stab incision through the right flank. Its inner end is placed behind the bile duct in the hepatorenal (Morison's) pouch.

Several points are important regarding drainage:

A drain is not inserted through a main wound but through a separate stab wound. The term is really a misnomer—small incision would be a better description.

There must be adequate exit for escape of bile or blood. If the drain tightly fits the opening in the abdominal wall it may well act as a cork. We have seen a huge collection of bile between the liver and the diaphragm despite the presence of a drain down to Morison's pouch, and suspect that the drain was blocking the drainage opening.

Closing the Wound. The following steps are taken:

1. The linea alba is closed with No. 40 linen sutures spaced a quarter inch apart. They are passed from deep to superficial, the knot thus being tied underneath (Fig. 8).

2. The peritoneum and the posterior sheath are closed with a running No. 1 plain catgut suture on a curved atraumatic needle, reinforced at every inch by a simple suture of the same type.

3. The anterior rectus sheath is closed by a running suture of No. 32 stainless steel wire.

4. The skin is closed with No. 60 linen sutures spaced half an inch apart. One or two of these pick up the anterior rectus sheath to obliterate the potential space between it and the skin.

5. Mastisol is painted round the wound and a gauze strip 12 layers thick is applied. A separate gauze dressing covers the drainage tube. A manytail bandage is put on.

After Treatment. Omnopon grain $\frac{1}{3}$ is given as required for pain. Pethidine overcomes spasm but is less effective for pain. The 5% glucose drip started on the table is discontinued when 1,000 c.c. have been given. The Levin tube is removed when the desire to vomit has passed. As many of these patients are stout and the right diaphragm moves poorly following operation, penicillin and streptomycin are given for a day or two. Deep breathing and limb movements are encouraged. The position in bed is changed 4-hourly. The patient dangles the legs over the side of the bed or gets up the day following operation.

The rule for the first few days is to walk or lie. To get a patient out of bed into an arm chair is to invite the very stasis of venous blood in the lower limbs which early rising is meant to avoid. The patient is encouraged to get to the toilet early. On the second evening following operation a mild aperient such as Agarol is given. No wound inspection is done in relation to any operation wound unless there is a positive indication for so doing. The dressings on the drainage tube are changed daily whilst there is any discharge. Stitches are removed on the eighth day and the drain at the same time.

It is wise to keep patients in an institution for a fortnight following any major operation. They are then under discipline which frequently does not obtain when they return home. Once in their accustomed surroundings they may be as active as they feel up to. They may drive a car after 3 weeks, but should not undertake the stresses of work or business until 6 weeks after surgery.

VARIATIONS IN THE OPERATION OF CHOLECYSTECTOMY

1. ACUTE CHOLECYSTITIS

When operation during an attack of acute cholecystitis was first popularized, it was widely practised and some surgeons are still enthusiastic about it.

It is based on the known fact that rupture of the inflamed and infected gall bladder is attended by a high mortality. In considering the question for and against operation in acute cholecystitis several facts should be considered:

(*a*) Acute cholecystitis is not analogous to acute appendicitis. In the latter, rupture is a common occurrence. It is very rare by comparison in the former.

(*b*) The acute gall bladder is never a 'middle of the night' emergency. This organ, if it does rupture, does not do so before the fifth day after onset of the attack.

(*c*) Rupture of the gall bladder is a disease of old people and the age of the patient is a factor to be considered.

(*d*) Although the removal of the acutely inflamed gall bladder may present no particular difficulty, it does happen that inflammation and oedema may increase the hazards of the operation or render this incomplete, so that cholecystectomy may have to be done. It may, for the same reasons, not be possible to deal with associated pathology of the bile ducts.

The matter should therefore be managed as follows: The patient is kept under institutional observation. If after 48 hours the patient's condition is getting worse, then operation is undertaken. There has been time for adequate preparation, hydration, etc. This method was practised in my wards and in private work and, almost without exception, the inflammation subsided and it became possible to plan operation at the optimum time (usually 6 weeks after the acute attack) and to work in an area devoid of the difficulties consequent on the presence of acute inflammation. Moreover, it is then possible to be more sure of anatomical relationships and to deal with the bile ducts and the choledochoduodenal sphincter.

The time, after commencement of the attack, when operation is most difficult is from about the tenth day. Although swelling and oedema are still in evidence, adhesions have become very dense and their separation entails danger to important structures. It thus becomes hazardous to expose the anatomy of the area.

2. THE FIBROSED GALL BLADDER

Not infrequently the gall bladder has been the site of long-continued chronic inflammation and its removal may be difficult. Dense fibrous tissue binds the gall bladder to neighbouring tissues. The risk of injury to important structures may be considerable.

The operation proceeds as follows:

(*a*) The first step is the separation of structures adherent to the gall bladder, viz. the omentum, the colon and the duodenum. Commencing at the fundus, adherent tissue is separated from the gall bladder, keeping the dissection in contact with this organ. There is risk of injury to the colon and especially to the duodenum. One type of duodenal diverticulum is due to a portion of the viscus being pulled up to the gall bladder by contraction of fibrous tissue resulting from pericholecystitis. If the bowel is opened, the hole is immediately repaired by a purse string suture, remembering that the outer layer should be of non-absorbable material such as cotton or linen, as the strong digestive juices of the duodenum may dissolve catgut.

The first part of the duodenum will be found drawn up to the porta hepatis and must be liberated and retracted downwards. At this stage of the operation the routine swabs and retractors are placed.

It is now possible to try to expose the cystic artery. If this is feasible, it is ligatured in continuity. Sometimes fibrosis in the duct area is so great that it is unwise to continue with the search. The surgeon then commences the removal of the gall bladder, beginning at the fundus. This route is now more than ever necessary because of the matting of tissues round the cystic duct and the obscuring of important anatomy. The peritoneum is so soldered to the gall bladder wall that no line of cleavage can be produced by trying to inject saline. It is then necessary to separate the liver from the gall bladder by making an incision where the two structures join and dissecting the liver off the gall bladder, keeping close to the latter. This is best done with a blunt instrument such a blunt-ended scissors or non-toothed dissecting forceps with blunt ends. The liver is very friable and can be deeply penetrated if the dissection is not maintained against the buried part of the gall bladder. On occasions the latter may have perforated into the liver and the gall bladder will then empty itself. Stones are picked up and bile or pus aspirated. The dissection may now be easier as the gall bladder can be grasped with ring forceps. The bleeding from the liver is easily controlled by placing a small swab in the cavity. There will be bleeding from branches of the cystic artery.

The dissection proceeds until the junction of the cystic duct with the main ducts is exposed. It may now be possible to tie the cystic artery and then the cystic duct and so complete the cholecystectomy.

INCOMPLETE CHOLECYSTECTOMY

It has been stated on authority times without number that the surgery of the bile ducts may be the most difficult of all types of surgery

This is borne out by the extensive series of reported cases, from many parts of the world, of injuries to the common bile duct.

Of cholecystectomy Lahey[1] has written:

'This is so trite that I blush every time I say it, but we ought to concentrate on giving more attention to publicizing how dangerous cholecystectomy is. We realize that cholecystostomy has been almost abolished, but thousands of cholecystectomies are being done every day and each one is a possible stricture. If the patient died instead of recovering after injury to his ducts, in many cases, he would be better off than to live with what he had to go through in the way of expense and suffering. We should publicize the fact that cholecystectomy is a dangerous operation. It is dangerous unless one realizes how important it is to control the blood supply, to demonstrate definitely the anatomic relationship, and to realize that anomalous anatomy is very common. I believe we could do nothing better than to write more and more on the need for accuracy in cholecystectomy'.

To circumvent the dire catastrophes attendant on gall bladder surgery, there have been laid down certain rules for guidance in the operation of cholecystectomy. These are mandatory if disaster is to be avoided. One of these laws is that the anatomy of the bile ducts must be clearly exposed before any structure is divided. It follows that in the absence of indications for duct exploration, such as stone or stricture, the procedure should be as follows:

The liberated gall bladder is split lengthwise, stones, etc. removed, and a lead probe pased into the cystic duct. With this guide it may be possible to continue the liberation further. Often this is inadvisable because of uncertainty about the position of essential structures. Sometimes no instrument will enter the duct. In such cases, having made sure by palpation that no stone is left in the cystic duct, the gall bladder is amputated up to that point to which it has been dissected free. Only then, as a rule, will the cystic artery be found indicating its presence by bleeding. The vessel is secured, usually by under-running with a No. 40 linen suture on a small needle. The mucous membrane in the small remaining stump may then be destroyed by a cautery or curetted away. The remaining wall of the gall bladder or duct remnant is sutured with non-absorbable interrupted sutures and a corrugated drain put into the hepato-renal (Morison's) pouch. The omentum or other fat is brought into contact with the stump, for in these cases the peritoneum is so involved in the fibrosis that it does not permit of mobilization.

In an experience extending over many years the author has utilized this procedure on many occasions. In none has there been cause for regret over the incomplete cholecystectomy, and in no case to the writer's knowledge has a further operation been necessary. Gall bladders may reform from remnants left *in situ* and cystic stumps have been reported as being the cause of recurrent symptoms, but no such complication has been met in the type of case described here. It is well recognized that the more severe the gall bladder pathology, the greater the benefit attendant on carefully planned operation.

3. Exploration of the Bile Duct

When exploration of the bile duct is indicated during a gall bladder operation, it is proceeded with as follows:

(*a*) The common duct has been visualized during the cholecystectomy. The duodenum is pushed distally with a Lahey swab on forceps. Gentleness is necessary, as the upper border of the genu of the duodenum is a vascular area. Two stay sutures of 000 plain catgut on a curved atraumatic needle are passed through the common duct about a ¼ inch apart, choosing an avascular area; mosquito forceps are applied to the stays. If the surgeon is uncertain whether he is dealing with a duct or a vessel, a hypodermic needle on a syringe is inserted and aspiration done. When assured

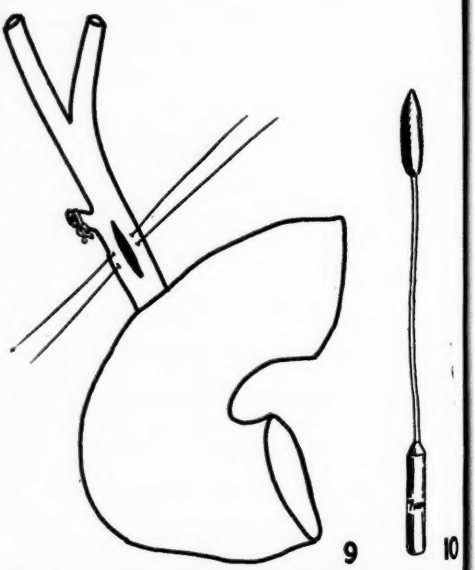


Fig. 9. Stay sutures distracting edges of opened bile duct.
Fig. 10. One of a series of malleable Baikie dilators.

on this matter a longitudinal slit ½ inch long is made between the stays (Fig. 9).

The character of the bile is noted. The field is kept clean by suction. Stones or debris are removed as they appear in the bile duct incision. A small catheter is then passed into the duct, which is forcibly irrigated with saline. This process is repeated several times both up and down. Desjardins forceps is passed into the duct and any abnormal content is gently extracted.

(*b*) The surgeon goes to the left side of the patient, retracts the second part of duodenum medially and with a knife divides the peritoneum to the right of the duodenum in a vertical direction. The area is avascular. The duodenum is then stripped medially, exposing the head of the pancreas and posterior to it the inferior vena cava. A lead probe is then passed into the bile duct and towards the duodenum. The surgeon is now able to palpate the whole length of the common duct and stones can be felt against the probe. Sometimes stones lie in a pocket of the duct. They are expressed and removed.

(*c*) *The Sphincter.* The operator holds the duodenum, the head of the pancreas and the bile duct in his left hand and with his right endeavours to pass the lead probe through the sphincter. Nice judgment is required to estimate a pressure which is not injurious to the duct. Should the probe not pass, it is removed and a Baike dilator inserted, bent to the necessary angle. A 4 mm. one is used first and smaller ones if necessary until the characteristic slip through the sphincter is felt by the hand passing the dilator. The left hand assists the passage by steadying the duodenum. The end of the dilator is then in the duodenum and can be pushed against its anterior wall to make quite sure of this (Fig. 10).

The theatre Sister now bends larger dilators to the size of the penetrating one and these are passed up to a size of 7 mm. This will not divulse the sphincter muscle. The duct is again irrigated, forcibly and often.

Sphincterotomy. Should it not be possible to negotiate the sphincter, the duodenum is opened. The papilla of Vater is situated more distally than one is apt to think.

First be sure that what appears to be duodenum *is* duodenum. It may be colon. Peritoneum passing from colon to duodenum is incised and the former is displaced distally. A lead probe in the common duct is made to push the posterior duodenal wall forward to indicate the site of the papilla. This known—two catgut stays are passed through duodenum: one at its right, the other at its left border and the bowel tensed between them. All coats are cut through boldly in a vertical direction and bleeders secured and tied with fine gut. The posterior wall of duodenum, pushed forward by the lead probe in the common duct is then inspected and the papilla sought. When found a probe is entered from the duodenal end, the bowel being steadied with stay sutures or Allis forceps. It should then be possible to pass a suitable Baikie from above. If the opening cannot be found, then a small incision is made on to the lead probe through the posterior duodenal wall. Once continuity of the duct with the duodenal lumen is established, a pair of small artery forceps is passed from the duodenum into the bile duct for ½ inch at 10 o'clock so that one jaw is in the lumen and the other on the posterior duodenal wall. The tissue between is crushed and the crushed area cut through with scissors (Figs. 11 A, B, and C).

The long limb of a T-tube is now passed from the bile duct into the duodenum. The

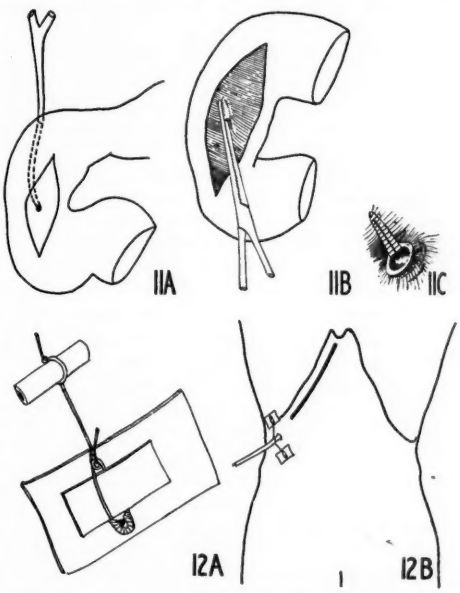


Fig. 11 A. The ampulla of Vater is shown through the the opening in the anterior wall of the duodenum.
Fig. 11 B. An artery forceps grips the wall of the sphincter at the 10 o'clock position.
Fig. 11 C. The artery forceps has been removed. The crushed tissue has been divided for 1 cm.
Fig. 12 A. The Lahey method of securing the T-tube by means of a safety pin and strapping.
Fig. 12 B. The operation completed.

incision in the anterior duodenal wall is then closed transversely by two layers of sutures. The first is an inverting 000 catgut all coats stitch. The second consists of interrupted mattress sutures of No. 60 linen thread. The bile duct incision is sutured round the emerging T-tube. A Penrose drain is put into the hepato-renal fossa, and is fixed to the skin by a loose stitch. The T-tube may be pulled out inadvertently in turning the patient. It is fixed by the method in use at the Lahey Clinic (Figs. 12 A and B).

Operative Cholangiography. It is a fairly usual practice to inject a contrast medium into the bile duct during operation. Having given the method a fairly extensive trial, it has been abandoned because it is not only time-taking but may also actually be misleading. Interpretation of the films is difficult and air bubbles may be mistaken for stones, etc. This is in agreement with authoritative opinion.

(*d*) *When and How to Drain the Common Duct:*

i. Should the exploration of the duct be negative, the incision in the duct is completely closed with 000 plain catgut.

ii. Should the duct have contained stones or debris, a No. 16 rubber catheter, with several holes in the end, is passed into the duct (whether up or down is of no significance) and fixed to the duct with a catgut stitch transfixing the catheter. The duct is snugly closed round this tube. Saline is injected into it to make sure it is watertight. It is a rule that before the operation is over the drain hole in bile duct must be watertight and bile must flow through the T-tube.

iii. Should the sphincter have been cut, or for any reason prolonged drainage of the common duct be decided on, a T-tube is inserted into the common duct. The upper limb need only be ½ inch long, the lower limb 1 inch unless it is decided to pass it into the duodenum, when it is left long enough for that purpose. This latter indication is rare. It may be necessary to keep the divided sphincter open until healing has occurred.

Note that any drain lying in the bile duct should not fit the tube tightly. When rubber is used anywhere in the body it is first inspected and stretched to ensure that it is 'live' rubber.

Removal of the drains. The drain in the hepato-renal pouch is removed when the stitches come out on the eighth day. The reason for this is that it is possible that a violent expulsive effort such as sneezing or coughing may dislodge the suture occluding the cystic duct. As the suture transfixes the duct it is more likely to be pushed off late than early and this may be followed by a copious outpouring of bile which may have a fatal result from bile peritonitis if there is no way to the exterior.

The T-tube is clamped off at night after two or three days. Vitamin K is given during the period of external bile drainage. On the twelfth post-operative day a contrast medium is injected through the T-tube. If this shows the common duct is still dilated, the T-tube is left *in situ.* If the duct is not dilated, the tube is gently pulled on each day until it comes out.

ACCIDENTS

(*a*) Injury to the liver may follow forcible retraction. If it occurs, a swab may be placed in the tear. As the venous pressure in the organ is low it will usually happen that the area has ceased to bleed by the end of the operation. If not, packing of the liver wound with Oxycel or Gelfoam is effective.

(*b*) Profuse bleeding may follow forcible retraction of the cystic artery, injury to an hepatic artery (the course of which is subject to wide variation) or damage to the portal vein. The cystic artery should not be tied together with the cystic duct (Lahey) (Figs. 13 A, B).

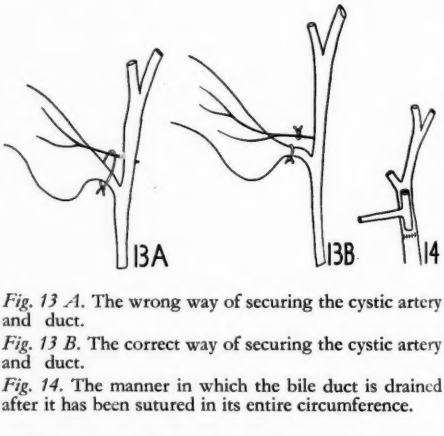


Fig. 13 A. The wrong way of securing the cystic artery and duct.

Fig. 13 B. The correct way of securing the cystic artery and duct.

Fig. 14. The manner in which the bile duct is drained after it has been sutured in its entire circumference.

The artery may be so pulled on by a common ligature that it is torn. The commonest cause of injury to the bile ducts occurs if profuse haemorrhage suddenly obscures the field and panicky action is taken. Endeavours to put artery forceps on a poorly visualized spurting vessel may quite well result in the bile

duct being caught in the grip of the forceps and ligatured. The proper procedure in the presence of profuse bleeding is for the surgeon to put the forefinger of the left hand in the foramen epiploicum (Winslow) and the thumb in front and compress the structures in the free border of the lesser omentum. This will immediately control all bleeding. It is then feasible to dry the field and expose and deal with the bleeder deliberately and safely. It is a good plan to keep a Blalock arterial clamp in the set and this may be put on, leaving the surgeon's hands free.

A cut in the hepatic artery is readily and securely closed by inserting one or more stitches with 00000 arterial silk.

(*c*) *Injury to the Common Duct.* This accident should not happen. If it does the very best time to repair the damage is at once. Should the duct be cut across it is immediately sutured with 000 plain catgut obtaining mucosa to mucosa apposition. Below this repair (or above, if necessary) the duct is opened by a small vertical incision and a T-tube put in, so that the repair is not vitiated by a tube passing across the suture line (Fig. 14).

OPSOMMING

In die tweede artikel van die reeks waarin mnr. A. Lee McGregor verkieslike operasies beskryf, bespreek hy chirurgie van die galblaas en die galbuise.

I wish to thank Dr. E. A. Thomas for the excellent drawings which illustrate the paper.

REFERENCE

1. Lahey, F. H. (1949): Ann. Surg., **129**, 6.

NOTES AND NEWS : BERIGTE

Mr. W. Girdwood, of Johannesburg, leaves for a short visit overseas on 15 April. He will spend some time in Professor Valdoni's Department of Surgery at the University of Rome.

He has been invited to lecture at the University of Innsbruck, where he will also do post-graduate work, attending surgical clinics and rounds.

Mr. Girdwood is accompanied by his wife. They will return to Johannesburg at the end of May.

* * *

Dr. R. Elsdon-Dew, Director of the Amoebiasis Research Unit of the Council for Scientific and Industrial Research, will be visiting the United States during May.

He has been asked to lecture at the University of California, Harvard University, Tulane University, Louisiana State University, and thereafter will be a member of the panel at the World Congress of Gastroenterology in Washington.

* * *

Dr. I. Freed, of Johannesburg, has been accepted as a post-graduate student to attend the *Course in Internal Medicine* given by Harvard Medical School for the months of June and July 1958, at the Peter Bent Brigham and the Massachusetts General Hospital.

Dr. Freed, accompanied by Mrs. Freed, will leave for Boston by air on 24 May, and return on 30 July 1958.

* * *

MEDICAL CONFERENCES IN 1958

American Goitre Association Conference—San Francisco	*April* or *May*
3rd World Congress of Gastroenterology — Washington, D.C.	*25–29 May*
8th Congress of the International Association for the Study of Bronchi—Munich	*16–17 May*
2nd World Congress of the International Federation of Obstetrics and Gynaecology —Montreal	*22–28 June*
Chest Conference (Commonwealth NAPT)—Incorporating the Annual Conference of the British Tuberculosis Association — Royal Festival Hall, London	*1– 4 July*
7th International Cancer Congress—London	*6–12 July*
3rd International Diabetes Congress—Dusseldorf	*21–25 July*
10th International Congress of Genetics—Montreal	*20–27 August*
General Assembly, World Medical Association—Copenhagen	*25–30 August*
International Dental Federation Conference—Brussels ...	*27 August–14 September*
4th International Congress of Biochemistry—Vienna	*1– 6 September*
5th International Congress on Diseases of the Chest—Tokyo	*7–11 September*
7th Congress of the International Society of Haematology—Rome	*8–13 September*
18th International Congress of Ophthalmology—Brussels ...	*8–12 September*
General Assembly of the International Organization Against Trachoma—Brussels	*8–15 September*
7th International Congress of Hydatidosis—Beirut	*12–15 September*
3rd World Congress of Cardiology—Brussels	*14–21 September*
3rd International Congress of Allergology—Paris	*19–26 October*
Neurological Surgeons Congress—San Francisco	*October* or *November*
7th International Congress of Leprosy—New Delhi	*8–14 December*

BUST OF SIR ALEXANDER FLEMING

PRESENTED TO THE WITWATERSRAND UNIVERSITY MEDICAL SCHOOL

At a ceremony held at the Waldorf Hotel, Johannesburg, on 28 February 1958, Mr. B. E. Bratt (Managing Director of Pfizer Laboratories South Africa (Pty.) Ltd.) presented a bust of Sir Alexander Fleming (the discoverer of penicillin) to the Witwatersrand Medical School.

The bust (chosen by Sir Alexander's brother, Mr. Robert Fleming, of London) was executed by the British sculptor Bevan.

In addition to members of the University, there were also representatives of the South African Institute for Medical Research, the South African Poliomyelitis Research Foundation, Baragwanath Hospital and the medical press.

Dr. H. J. de Graaf (Acting Head of the Department of Pharmacology) received the bust in the presence of the Principal and Vice-Chancellor of the University, Prof. W. G. Sutton.

The bust will probably be placed in the foyer of the Medical School, Hospital Street, Johannesburg.

Dr. H. J. de Graaf (*Acting Head of the Department of Pharmacology*), Mr. B. E. Bratt (*Managing Director of Pfizer Laboratories South Africa (Pty.) Ltd.*) and (on the extreme right) Prof. W. G. Sutton (*Principal and Vice-Chancellor of the University*).

* * *

MEDICAL FILMS

Glaxo Laboratories (Pty.) Ltd. have established a library of medical films. These films are available to any groups of doctors who may be interested in borrowing them. The first 3 films are all 16 mm., in colour and with a sound track. The details are as follows:

1. *Food for Thought* (596 feet), running time 15 minutes. A nutritional research team plans a complete food—*Complan*.
2. *A Vitamin Emerges* (560 feet), running time 15 minutes. How Vitamin B12 was isolated at Glaxo Laboratories and how research is increasing our knowledge of this vitamin.
3. *Longer Life for BCG* (555 feet), running time 15 minutes. The contribution of research to the manufacture and standardization of freeze-dried BCG vaccine.

Readers interested in borrowing these films should communicate with Glaxo Laboratories (Pty.) Ltd., P.O. Box 21, Wadeville, Transvaal.

As further films are added to the list, they will be announced in these columns from time to time.

PREPARATE EN TOESTELLE

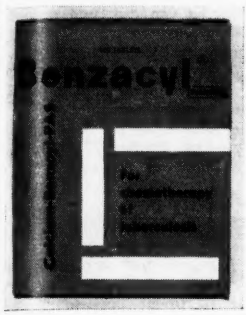
BENZACYL

Benzacyl is 'n PAS-derivaat wat byna geeneen van die nadele van die oorspronklike produk openbaar nie. Net soos PAS is dit saamgestel vir die gesamentlike langtermynbehandeling van tuberkulose.

Benzacyl is die kalsiumsout van N-bensoïel-p-aminosalisielsuur (kalsium-bensoïel-PAS, Benzopas of BPAS). Daar is aanvanklik gemeen dat dit sy effek uitoefen deur PAS stadig in die liggaam vry te stel, maar dit skyn nou dat dit nie die geval is nie. Dit word aangetoon deur die feit dat *Benzacyl* wat byna geheel en al uit die ingewande geabsorbeer word, saam met die urine afgeskei word grotendeels in die vorm van bensoïel-PAS, met slegs klein hoeveelhede PAS, geasetileerde PAS en geglikokoleerde PAS.

Die werklike werkwyse van *Benzacyl* is nie bekend nie. Dit verskil duidelik van dié van PAS.

want dit word nie onaktief gemaak deur hoë konsentrasies para-aminobensoësuur nie. *In vitro* is *Benzacyl* 100 keer minder aktief as PAS, maar *in vivo* is die 2 produkte, gewig vir gewig, ewe doeltreffend. In die praktyk lewer *Benzacyl* egter beter resultate op, want dit besit die volgende belangrike voordele in vergelyking met PAS:



1. *Doeltreffende Bloedpeile word Makliker in Stand Gehou: Benzacyl* word stadig geabsorbeer en stadig afgeskei (spitsbloedpeile word eers 3 uur na opneming bereik). Derhalwe hoef dit slegs 3 maal per dag geneem te word. PAS wat vinnig geabsorbeer en vinnig afgeskei word, behoort teoreties al om die 3 uur dwarsdeur die dag en nag toegedien te word. Aangesien dit in die praktyk selde gebeur, word standhoudende bloedpeile nie met PAS nie, maar wel met *Benzacyl* in stand gehou.

2. *Beter Absorpsie. Benzacyl*-tablette desintegreer byna oombliklik in water, en volledige absorpsie word derhalwe verseker. PAS-tablette het veel meer tyd nodig om te desintegreer. Trouens, gevalle is bekend waar die tablette dwarsdeur die liggaam gegaan en heel afgeskei is.

3. *Word Beter Verdra. Benzacyl* veroorsaak nie soveel versteurings van die maag soos PAS nie. Daar is min pasiënte wie se behandeling ten gevolge van maagkwale stopgesit moet word. Daarbenewens is twee-derdes van die pasiënte wat PAS nie kan verdra nie, in staat om *Benzacyl* sonder enige moeilikheid te neem.

4. *Dit Smaak Beter. Benzacyl* het feitlik niks van die onaangename smaak wat so baie pasiënte laat weier om PAS te neem nie.

5. *Geriefliker.* Vier *Benzacyl*-tablette hoef in die gemiddelde geval slegs 3 maal per dag toegedien te word, in vergelyking met groter dosisse PAS wat meer dikwels geneem moet word.

6. *Minder Weerstand.* Weerstand teen *Benzacyl* word nie so maklik opgebou soos teen PAS nie. Daar is geen kruisweerstand tussen die 2 middels nie. *Benzacyl* kan dus van groot waarde wees vir pasiënte wat reeds weerstand teen PAS opgebou het.

7. *Minder Allergiese Reaksies.* Hulle kom selde voor met *Benzacyl*. Daarenteen word bereken dat 4% van alle gevalle wat met PAS behandel word, sodanige reaksies openbaar.

Met die oog op hierdie voordele neem *Benzacyl* geleidelik die plek in van PAS in baie lande van die wêreld. *Benzacyl* is vir die eerste keer in 1948 beskikbaar gestel, en die waarde daarvan is sederdien bevestig deur talle navorsingswerkers wat die mening toegedaan is dat *Benzacyl* nie alleen vir 'n hoër persentasie genesings as PAS sorg nie, maar ook die tyd wat die tuberkulose-pasiënt in die hospitaal moet deurbring, verkort.

Benzacyl is verkrygbaar in bottels van 400 en 2,000 tablette van 1.0 g., en in bottels van 100 sakkies, elk bevattende 4.0 g. *Benzacyl*-poeier, van die alleenagente vir Suid-Afrika:

Westdene Products (Pty.) Ltd., Essanby-gebou 23, Jeppestraat 175, Johannesburg, of van die firma se takke in Kaapstad, Durban of Pretoria.

'N VERSTELBARE BOYLE-TONGPLAAT

VIR PASIËNTE VAN ENIGE OUDERDOM

Westdene Products (Pty.) Ltd., van Essanby-gebou 23, Jeppestraat, Johannesburg, kondig die beskikbaarstelling aan (Fig. 1) van Magauran se Aanpasser (A) en Mobiele Buisspatel (B), vervaardig deur Dwn Bros., Mayer & Phelps, Ltd.

Die doel van die toestel is om 'n Boyle-tongplaat beskikbaar te stel wat aangepas kan word by pasiënte van enige grootte—vanaf die klein kind tot die groot volwassene.

'n Staalbuis steek aan die distale kant van die spatel uit, en hieraan kan 'n intratrachea-buis bevestig word. Die klein plaatjie (C) wat oor die buisspatel skuif, word gebruik om die spatel vir buitengewoon groot pasiënte te verbreed. Die aanpasser

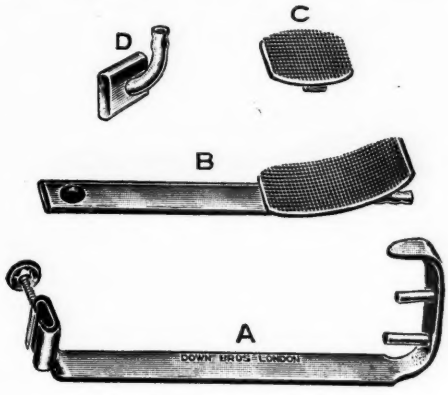


Fig. 1

kan op so 'n manier gesuspendeer word dat die geneesheer die buisspatel teen verskillende hoeke kan gebruik.

Die toestel kan gebruik word met Draffin se tweepoot of die Magauran-driehoekraam, soos verduidelik in die *Lancet*, 9 November 1957, op bl. 927.

KUNSMATIGE ASEMHALING

Westdene Products (Pty.) Ltd. kondig die aankoms van twee nuwe reddingstoestelle aan.

1. Die *Ambu*-bewussynhersteller (Testa-Laboratorium, Kopenhagen), 'n self-opblasende sak-bewussynhersteller wat met die hand werk.

2. Die *Ambu*-voetsuigpomp (Testa-Laboratorium, Kopenhagen).

Hier is twee *nuwe* reddingstoestelle wat oral en te enigertyd gereed vir gebruik is omdat hulle nie van suurstofsilinders of waterdruk afhanklik is nie.

Omdat voorbereiding vir gebruik en alle uitwendige kragbronne uitgeskakel is, kan die toestelle meer lewens red omdat hulle waardevolle tyd bespaar.

Die *Ambu*-bewussynhersteller kan in oomblikke van nood gebruik word sonder enige verbindings, of die noodsaaklikheid om meters te stel. Die eenheid is klein, kompak en lig. Dit bied u vingertoppbeheer, en 'n mens kan ure lank daarmee werk sonder om moeg te word. Die sak verskaf 400 tot 1,300 ml. lug met 'n enkele kompressie, en *hou die fisiologiese korrekte ritme in stand* ten gevolge van sy self-opblasende konstruksie. Die *Ambu*-bewussynhersteller is voorsien van 'n spesiale klep vir suurstofverrykte mengsels, indien dit nodig word om sulke mengsels te gebruik.

Die *Ambu*-suigpomp wat met die voet werk, verskaf negatiewe druk van 200 mm. tot 300 mm. Hg. Die ligte eenheid werk sonder elektriese motors of waterdruk en *sal voortgaan om te werk met 'n vol bottel*, of heeltemal sonder 'n bottel.

Die opvallendste kenmerke van die twee eenhede is:

i. Die eenvoudige werkverrigting;
ii. Die sterk konstruksie;
iii. Die vermoë om te werk waar ook al kunsmatige asemhaling toegepas moet word.

Alleeninvoerders en -Verspreiders vir Suid-Afrika: Westdene Products (Pty.) Ltd., Posbus 7710, Johannesburg

PREPARATIONS AND APPLIANCES

BENZACYL

Benzacyl is a PAS derivative which is almost free from the disadvantages of the original product. Like PAS, it is intended for the combined long-term treatment of tuberculosis.

Benzacyl is the calcium salt of N-benzoyl-p-aminosalicylic acid (calcium benzoyl-PAS, Benzopas or BPAS). It was first thought that it acted by slowly releasing PAS in the body, but is now appears that this is not the case. This is shown by the fact that *Benzacyl*, which is almost completely absorbed from the bowel, is excreted in the urine largely as benzoyl-PAS with only small quantities of PAS, acetylated PAS and glycocolated PAS.

The actual mode of action of *Benzacyl* is not known. It clearly differs from that of PAS because it is not inactivated by high concentrations of para-aminobenzoic acid. *In vitro Benzacyl* is 100 times less active than PAS but *in vivo* the 2 products are weight for weight equally effective. In actual practice, however, *Benzacyl* gives far better results because it has the following distinct advantages over PAS:

1. *Adequate Blood Levels are more easily Maintained: Benzacyl* is slowly absorbed and slowly excreted (peak blood levels are not reached until 3 hours after ingestion). Therefore it need be given only 3 times a day. PAS, because of its rapid absorption and excretion should theoretically be given every 3 hours round the clock. Since in practice this is rarely done, consistent blood levels are not maintained with PAS as in the case of *Benzacyl*.

2. *Better Absorption: Benzacyl* tablets disintegrate almost immediately in water and complete absorption is therefore assured. PAS tablets have a much longer disintegration rate and in fact cases have been reported of the tablets passing right through the system and being excreted whole.

3. *Better Tolerated: Benzacyl* does not cause gastric upset to the same extent as PAS. Few patients are unable to continue treatment because of this trouble. In addition two thirds of the patients who cannot tolerate PAS are able to take *Benzacyl* without difficulty.

4. *Better Taste: Benzacyl* is almost free from the most unpleasant taste which makes many patients refuse PAS.

5. *Greater Convenience:* Four *Benzacyl* tablets need be given only 3 time a day in an average case, compared with the higher and more frequent dosage of PAS.

6. *Less Resistance:* Resistance does not build up as readily to *Benzacyl* as to PAS. There is no cross resistance between the 2 drugs so that *Benzacyl* may be useful in cases which have developed resistance to PAS.

7. *Fewer Allergic Reactions:* They rarely occur with *Benzacyl* as compared with the estimate of 4% of all cases treated with PAS.

Because of these advantages, *Benzacyl* is gradually replacing PAS in many countries throughout the world. Originally introduced in 1948, its value has now been confirmed by many workers who suggest that Benzacyl will not only give a higher percentage of cures than PAS but will also reduce the hospitalization time for tuberculosis patients.

Benzacyl is available in bottles of 400 or 2,000 tablets 1.0 g. and in bottles of 100 sachets each containing 4.0 g. *Benzacyl* Powder, from the sole South African Agents: Westdene Products (Pty.) Ltd., 23 Essanby House, 175 Jeppe Street, Johannesburg or from the Cape Town, Durban or Pretoria branches.

ADJUSTABLE BOYLE'S TONGUE PLATE

FOR PATIENTS OF ANY AGE

Westdene Products (Pty.) Ltd., of 23 Essanby House, Jeppe Street, Johannesburg, now have available (Fig. 1) Magauran's Adaptor (A) and Mobile Tube Spatula (B), manufactured by Down Bros., Mayer & Phelps Ltd.

The object of the device is to provide a Boyle's tongue plate which can be adjusted to suit patients of any size, from a small child to a big adult.

From the distal part of the spatula a steel tube projects, to which a short intratracheal tube can be attached. The small plate (C), which slides on to

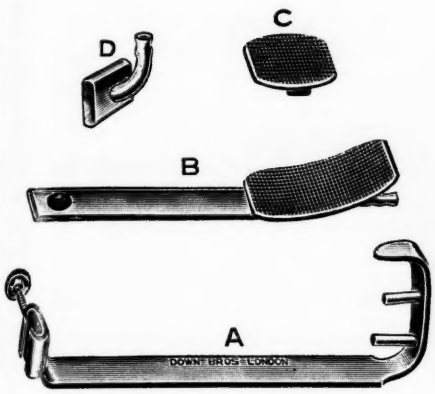


Fig. 1

the tube spatula, is used to widen the spatula for outsize patients. The adaptor can be suspended in such a manner as to give varying degrees of tilt to the tube spatula.

The device can be used with Draffin's bipods, or the Magauran triangular frame as mentioned in the *Lancet*, 9 November 1957, at p. 927.

ARTIFICIAL RESPIRATION

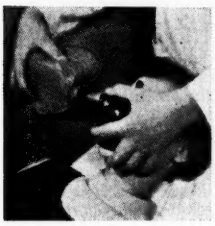
Westdene Products (Pty.) Ltd. announce the arrival of *two new life-saving aids*:

1. The *Ambu* Resuscitator (Testa-Laboratorium, Copenhagen), a manually operated, self-inflating bag resuscitator.

2. The *Ambu* Foot-Suction Pump (Testa-Laboratorium, Copenhagen).

Here are two *new* pieces of life-saving equipment which are ready for use anywhere and any time, because they do not require oxygen cylinders or water pressure.

Because preparation for use and exterior power source have been eliminated, the equipment *can save more lives by saving valuable time.*



The *Ambu* Resuscitator can be used at the moment of emergency without need of connexions or setting of gauges. The unit is small, compact and light. It provides finger-tip control and can be operated for hours without tiring the operator. The bag provides 400 to 1,300 ml. of air with one compression and *maintains a physiologically correct rhythm* through its self-inflating construction. A special valve is fitted to the *Ambu* Resuscitator which provides for oxygen-enriched mixtures, if so desired.

The foot-operated *Ambu* Suction Pump provides negative pressure of 280 mm. to 300 mm. Hg. The light-weight unit operates without electric motors or water pressure and *will continue operating with a full bottle* or without any bottle at all.

The outstanding advantages of these 2 units are:

i. Simplicity of operation;

ii. Rugged construction;

iii. Ability to work *wherever* respiratory failures occur.

Sole South African Importers and Distributors: Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

REVIEWS OF BOOKS

PORTAL HYPERTENSION

A Contribution to the Study of Portal Hypertension. By Alan Henderson Hunt, M.A., D.M., M.Ch., F.R.C.S. (Jacksonian Prize Essay, Royal College of Surgeons 1956). 1958. (Pp. 224 + Index. With 120 Figs. 40s.). Edinburgh and London: E. & S. Livingstone Ltd.

Portal hypertension is a subject which, of recent years, has been of interest to the surgeon because surgical measures offer the best hope of relief from the complications of the condition.

This publication (which embodies the content of a Jacksonian essay) is not intended to be a comprehensive treatise on portal hypertension. Nevertheless, it covers adequately all the requirements for the management of the condition from a surgical standpoint. The volume contains many useful practical suggestions which can only have been made on the basis of an extensive experience of portal hypertension.

The author analyses 250 personal cases of portal hypertension and the results of surgery have been assessed on 69 cases of porta-caval and 37 cases of lieno-renal anastomosis.

Mr. Hunt emphasizes the need to know the anatomy of the individual portal vein in each case and stresses the venous and arterial anomalies which may seriously impair the performance or the result of an operation.

Knowledge of the portal vein is derived from splenic or portal venography and there is an excellent account of variations of the portal vein as seen with this type of investigation. It appears that this is probably the most essential investigation in these cases.

The factors causing extra- and intra-hepatic portal obstruction are succinctly discussed and are followed by a concise account of the positions and nature of the abnormal communicating channels in portal hypertension.

No attempt is made to discuss the pathology of the liver conditions.

Portal stasis as distinct from portal pressure is important in treatment. Alterations in the pressure readings seem to be of no prognostic significance, so far as operative procedures are concerned. On the other hand, an increase in the blood velocity in the portal vein occurs in a successful anastomosis.

The author has set out clearly the clinical manifestations, the investigation of a case of portal

hypertension and the indications for operation, based upon the findings in his series of patients.

Since the operation is likely to be of a major nature, the pre-operative preparation of the patient is stressed and fully described and the management of hepatic coma is emphasized. Both the emergency treatment of the complications of portal hypertension and the elective treatment of the condition are considered in some detail, while the haemodynamic effects of portal systemic anastomosis and the complications of the operations are fully discussed.

The results in this series indicate that one of the shunting procedures is likely to be the most suitable in the treatment of portal hypertension.

The details of the technical considerations are set out in a number of *Appendices* which cover such subjects as the position of the patient on the operating table, splenic venography, taking of portal pressure, portal venography, measurement of speed of blood flow with the double scintillation detector, anaesthesia, technique of porta-caval and lieno-renal anastomosis, gastric transection and resection, oesophagotomy and ligature of oesophageal varices. The final *Appendix* consists of the case histories of 100 cases of portal hypertension.

This is a book for the specialist. Each chapter contains much of value to the surgeon interested in portal hypertension and the value is enhanced by the excellence of the photographs and the presentation of the material.

Mr. Hunt has made a contribution to the knowledge of portal hypertension by collating all his findings within the covers of a single monograph.

MEDICAL PROGRESS

The British Encyclopaedia of Medical Practice. Including Medicine, Surgery, Obstetrics, Gynaecology and other Special Subjects: Medical Progress 1957. Editor in Chief: The Lord Cohen of Birkenhead, M.D., D.Sc., LL.D., F.R.C.P., F.A.C.P., F.F.R. (Pp. 339 + Index). London: Butterworth & Co. (Publishers), Ltd.

This addendum to the *British Encyclopaedia of Medical Practice* summarizes medical advances during 1957. It is divided into the three parts which have become typical of these annual supplements, viz. *Part I—Critical Surveys*; *Part II—Drugs* and *Part III—Abstracts*.

The review of advances in medical genetics forms a very important section of this volume. In recent years there has been a growing conviction that peptic ulcer occurs much more commonly in group O persons. Indeed, Aird *et al.* expressed the view that persons of group O were 35% more likely to develop peptic ulceration than were persons of other groups. It is, however, interesting that when this hypothesis was tested with adequate controls, it was found that peptic (duodenal) ulcer did not occur significantly more often in association with group O. This little frolic in the study of aetiology should emphasize the suspicion with which statistical generalizations must be regarded.

The postulated increase in the mutation rate as result of exposure to increased irradiation is also reviewed. There is obviously much assumption and little proof which underlies the totally uninhibited speculation typical of discussion in this field. It should not be forgotten that an analysis of 80,000 pregnancies in Hiroshima and Nagasaki between 1946 and 1953 provided no evidence of genetic or other damage in the first generation.

There is some evidence, however, of somatic cell damage due to irradiation, e.g. an increased leukaemia rate following the atomic explosions over Japan, as well as an increase in the incidence of microcephaly in children exposed *in utero* to the atomic explosion.

The following list of *Critical Surveys* indicates the very comprehensive scope of the advances summarized in this volume: Medicine (*Smart*); Surgery (*Edwards*); Obstetrics and Gynaecology (*Bender*); Cardiovascular Diseases in Children (*Hay*); Epilepsy (*Hill*); Malignant Disease (*Cade and Windeyer*); Medical Genetics (*Clarke*); Rheumatology (*Copeman and Savage*); Tropical Medicine (*Adams*) and Vitamins (*Harris*).

ANAESTHETIC ACCIDENTS

Anaesthetic Accidents: The Complications of General and Regional Anaesthesia. By V. Keating, M.B., B.Ch., D.A., F.F.A.R.C.S. 1956. (Pp. 261 + vi. 31s. 6d.). London: Lloyd-Luke (Medical Books) Limited. *Sole Distributors for Southern Africa*: P. B. Mayer, P.O. Box 713, Cape Town.

The author's experience of the problems of anaesthesia, gathered in different parts of the world under a variety of climatic conditions, has equipped him to deal with the numerous practical problems which are a feature of anaesthetic practice.

There is an adequate physiological introduction which is essential for an intelligent grasp of the principles of anaesthesia. This, coupled with a sound knowledge of the pharmacology of the anaesthetic drugs in common use, is the surest protection for the anaesthetist against unexpected accidents. One of the most tragic catastrophies which can befall the anaesthetist is the administration of the incorrect drug or gas.

Dr. Keating devotes a special chapter to safety precautions and emphasizes the need to form the habit of 'trusting *no one* who attaches cylinders to a gas machine . . .' (p. 249). He takes the view that unless there is a high index of suspicion on the part of the anaesthetist, accidents due to the use of the wrong gas will continue, and recommends the system employed in the United States to prevent disasters of this kind. An obvious and certain stratagem is to make it mechanically impossible to connect wrongly any link in the chain which may stretch from a bank in the basement to an operating theatre on a remote floor. It is impossible entirely to eliminate human frailty, and this is a field in which caution and precaution cannot be excessive.

Dr. Keating concludes his volume with a short chapter on *Medico-Legal Considerations* in which he discusses the important concept of negligence. He emphasizes that (p. 251) 'the possession of a specialist diploma does not in itself mean that the possessor has the requisite degree of skill, nor does its absence mean that he has not'. He also points out, quite rightly, that it is rare for the pathologist to be able to assess the cause of death precisely in fatal anaesthetic accidents: '. . . the absence of definite disease does *not* indicate bad management by the anaesthetist. Death in the theatre, with no ascertainable cause, was not uncommon before the days of anaesthesia' (p. 253).